The New England
Journal of Medicine

VOLUME 213 AUGUST 1, 1935 NUMBER 5

The Massachusetts Medical Society

SECTION OF PEDIATRICS

Georgian Room, Hotel Statler, Boston, June 5, 1935, 9 A.M.

PRESIDING:
Dr. Joseph Garland, Chairman.
Dr. James M. Baty, Secretary.

SYMPOSIUM ON THE CONTROL OF CERTAIN COMMUNICABLE DISEASES

CHAIRMAN GARLAND: The meeting will please come to order. One of the first duties in opening the meeting is to appoint a nominating committee to furnish names of officers for the next year. I will appoint Dr. Fred Allen of Holyoke, as Chairman, Dr. John Lovett Morse and Dr. Elmer Barron.

It has seemed fitting to the officers of the Section this year in view of the new procedures that have been developed along epidemiological lines, to have a symposium which would help to clarify some of these rather difficult problems, and with that end in view we have, I think, arranged a valuable and interesting program.

The first paper on this program, which we are calling a Symposium on the Control of Certain Communicable Diseases, will be a paper on measles control by Dr. R. Cannon Eley of the Children's Hospital.

THE CONTROL OF MEASLES*

BY R. CANNON ELEY, M.D.†

THE control of measles by public health measures such as placarding, quarantine, etc., is inadequate; nor does there exist at the present time a satisfactory method for active immunization against the disease. However, that measles may be either modified or prevented after an individual has been exposed, by the administration of human immune antibodies, has been satisfactorily established. Furthermore, the advisability of either modifying or preventing the occurrence of this disease in certain individuals, and particularly among infants and children, has become recognized as a warranted procedure in the practice of medicine. Therefore the problem concerned in the control of this disease resolves itself into three phases, namely, the epidemiological factors concerned in the transmission and dissemination of the disease, the selection of the proper patients for modification or prevention, and finally, the selection of a satisfactory method for the production of passive immunization. The first phase is well understood and does not warrant consideration at this time. In the following discussion I shall confine my remarks to the consideration of the last two aspects, namely, the selection of patients for treatment and the various prophylactic measures that may be employed in the modification or prevention of this disease.

The selection of patients for modification or prevention of measles necessitates not only the consideration of the individual, but also a critical appraisal of the circumstances which resulted in the patient's exposure to the disease. In considering the patient the first question to be entertained is whether prophylactic measures should be employed to prevent or to modify the infection. The solution to this problem depends upon the physical condition of the individual, as measles and its complications should be avoided in chronically ill, debilitated, tuberculous and acutely ill children. However, if the patient is in good health and the accompanying circumstances do not contraindicate the procedure, efforts should be made to obtain modification, as complications are infrequently encountered with this form of the disease. Furthermore it is generally believed that permanent immunity may result from the modified form.

The circumstances which resulted in the exposure of the patient may, however, be of such a nature as to make protection rather than modification desirable. Thus in institutions such as foundling asylums, orphanages, and sanatoria it would be advisable to institute prophylactic

*From the Department of Pediatrics, Harvard Medical School, the Department of Communicable Diseases, Harvard School of Public Health, and the Children's and Infants' Hospitals, Boston.

Read before the Section of Pediatrics at the One Hundred and Fifty-Fourth Annual Meeting of the Massachusetts Medical Society, Boston, June 5, 1935, as part of a symposium on the control of certain communicable diseases.

†Eley, R. Cannon—Associate in Pediatrics and Communicable Diseases, Harvard Medical School and Harvard School of Public Health. For record and address of author see "This Week's Issue," page 240.

measures as soon after the exposure as possible in order to eradicate the disease immediately. As the immunity conferred by the usual prophylactic measures is of short duration (four to five weeks), the converse of this is true when the disease occurs in epidemic proportions outside of institutions, for in such situations modification with resultant immunity (thereby gradually terminating the epidemic) is to be sought. If this is not obtained, individuals will repeatedly be reëxposed necessitating further prophylactic treatment.

At the present time passive immunization may be obtained by the administration of convalescent serum, adult immune serum, adult immune whole blood and immune globulin obtained from human placentae. As can be seen in table 1 the effectiveness of each of these procedures depends upon such factors as degree of antibody content, dosage, time of administration in regard to exposure, type of exposure, and the age and size of the patient. Therefore in considering the merits of each of these prophylactic measures care must be exercised that the above-named factors are duly considered.

TABLE 1

PASSIVE IMMUNIZATION

Method	Dosage
Convalescent Serum	4-6 cc.
Adult Immune Serum	15-20 cc.
Adult Immune Whole Blood	30-40 cc.
Placental Extract	2-6 cc.

Factors Influencing Passive Immunization

1. Potency of material employed
2. Dosage
3. Time of administration
4. Age of patient
5. Degree of exposure

Table 2, which has been compiled from statistics available in the literature[1, 2, 3, 4, 5, 6], well illustrates the influence that the above-mentioned factors may exert upon the results obtained by any of the prophylactic measures. (In order to simplify the table the age groups and time of treatment have been omitted. The ages ranged from six months to fifteen years. All patients were treated within seven days or less after exposure.) Thus it can be seen that the percentage of successful treatments obtained by convalescent serum was almost twice that obtained by the use of adult immune serum even though the dosage employed was much less. In fact, in order to obtain the same percentage of protection with adult serum as with convalescent serum, from three to four times the dosage was necessary. The marked difference between the successful protections obtained with patients treated on account of exposure in the hospital as compared with the group treated for exposure at home is not surprising for undoubtedly many of the patients considered as exposed in the hospital probably were not infected at all. This would be particularly applicable in those institutions that employ the so-called cubicle

TABLE 2

Author	Serum	Cases		Protected	Modified	Failed
1. Schick and Karelitz	Hosp. A. S. 6 cc.	69		83%	17%	0
	Home 6 cc.	65		29%	22%	49%
	8 cc.	75		46%	43%	11%
	12-25 cc.	54		57%	20%	23%
2. Morales and Mandry	Home A. S. 10 cc.	138		41%		
	15-30 cc.	321		58%	22%	20%
	30 cc.	34		82%		
	40 cc.	28		82%		
	Conval. serum 4-6 cc.	120		84%	11%	5%
3. Park and Freeman	Conval. serum	652	Hosp.	91%	7%	2%
	Conval. serum 6 cc.	226	Home	52%	42%	6%
4. Levinson et al.	Conval. serum 3 cc. / 3 yrs. plus 0.5 cc. per 6 mos.	287	Home	60%	33%	7%
5. Nabarro and Signy	Conval. serum 5-7 cc.	461	Hosp.	97%	——	3%
	Conval. serum	125	Home	94%	——	6%
6. McKhann et al.	Immune Globulin	1341	Both	73.3%	23.4%	3.2%

A. S. = Adult serum
Conval. serum = Convalescent serum

system or where isolation technique is enforced. That an increase in the percentage of patients protected was obtained as the dosage of the serum employed was increased was to be expected as there was a proportionate increase in the antibody content. This fact illustrates the uncertainties encountered in the employment of either adult or convalescent serum when the potency is unknown. For the purpose of contrast the patients treated with immune globulins (obtained from human placental extract) were not subdivided in this table into those exposed at home and those exposed in hospitals. Instead all of the patients, regardless of the type of exposure, were grouped together; of 1341 patients treated 73.3 per cent were completely protected; 23.4 per cent had a modified form of measles, and failure occurred in only 3.2 per cent. In all instances a standard dose of 5 cc. was employed. The practicable value of a uniform dose does not need comment.

Table 3 demonstrates the results obtained when individuals, who were *intimately exposed,* were treated with placental extract in an effort to obtain either complete protection or modification. If these figures are compared with those of Morales and Mandry, Park and Freeman, Levinson, and Nabarro and Signy (all patients received convalescent serum within five days or less after exposure and therefore should have obtained either modification or protection) it will be seen that the results obtained with the two procedures are comparable. This would indicate that placental extract is as effective as convalescent serum when employed in the prophylactic treatment of measles. This observation is further substantiated by table 4 which shows that although the percentage of patients *completely protected* was greater in the group treated with convalescent serum than with placental extract, yet the percentage of *failures* was less with placental extract than with convalescent serum.

TABLE 3

INTIMATE EXPOSURE

Solution (or Fraction)	Given to Protect				Given to Modify			
	Cases	P.	M.	F.	Cases	P.	M.	F.
T (0-50)	28	22	6	0	49	18	27	4
M (28-50)	47	26	14	7	152	67	69	16
R (28-50)S	16	13	3	0	19	12	5	2
S (0-50)	31	23	7	1	66	20	43	3
P Iso. P.	16	5	9	2	36	9	21	6
FP	9	2	6	1	13	5	8	0
V (S-TG)	3	3	0	0	14	5	9	0
W (T-TG)	0	0	0	0	6	4	2	0
Commercial Extract	24	12	11	1	102	68	30	4
Total	174	106	56	12	457	208	214	35
%		60.9	32.2	6.9		45.5	46.8	7.7
%		93.1				92.3		

P—protected
M—modified
F—failed

One of the important questions that must be considered when any therapeutic measure is being employed is the possible untoward effects of the procedure upon the patient. Such manifestations, which usually are referred to as reactions, may be local or systemic in nature and have been noted following the institution of prophylactic therapy in measles. The fact that convalescent serum usually may be injected in adequate therapeutic amounts with only slight, if any, local or systemic reaction, has made this method superior to the employment of adult immune serum or adult immune whole blood, for when these substances are administered in amounts adequate to obtain therapeutic results, an area of tenderness, edema, and discoloration not infrequently develops at the site of the injection. Whether a systemic reaction, which usually manifests itself by malaise, indisposition, anorexia and temperature, will accompany this local reaction, largely depends upon the degree of the local disturbance.

TABLE 4

PATIENTS TREATED FOR PROTECTION OR MODIFICATION

All Types of Exposure

Procedure	No. Cases	Protected		Modified		Failed	
Adult Serum	584	329	56.4%	139	23.8%	116	19.8%
Conv. Serum	1627	1227	75.4%	273	16.8%	127	7.8%
Plac. Extract	1341	959	71.5%	321	23.9%	61	4.6%

Local and systemic reactions have been observed following the intramuscular administration of placental extract. As one might expect, these reactions occurred with greater frequency

during that period of time when the extract was first being employed and when some confusion naturally existed as to which of the various protein fractions possessed the immune bodies. Table 5 illustrates this point. In this table an effort has been made to tabulate all of the reactions, regardless of how mild or how transient, that have been noted following the administration of the extract. If the patient suffered the slightest pain or showed the slightest discoloration at the site of injection, a local reaction was recorded. This type of reaction occurred in 25.3 per cent of 1232 patients; 151 patients, or 12.3 per cent, presented a febrile reaction of one degree or less; a more intense local reaction appeared in 4 per cent of the group and only thirty-one patients or 2.5 per cent showed a febrile reaction of 101° or more. From these statistics it would appear that neither the frequency nor the severity of the reactions are sufficient to contraindicate the use of placental extract as a prophylactic measure in the control of a disease as serious as measles.

TABLE 5

REACTIONS

Fraction	Total No. Patients	No Reaction		Local		Febrile		Local ++		Febrile (T = 101+)	
T	239	151	63.2%	58	24.3%	41	17.1%	6	2.5%	9	3.8%
M	436	317	72.7%	99	22.7%	35	8. %	8	1.8%	4	.9%
R	144	112	77.7%	21	14.6%	14	9.7%	1	.7%	2	1.4%
S	248	172	69. %	69	27.8%	20	8.1%	11	4.4%	4	1.6%
P	137	71	51.8%	56	40.9%	32	23.4%	10	7.3%	5	3.6%
V	22	5	22.7%	7	31.8%	4	18.2%	10	45.5%	7	31.8%
W	6	1	16.7%	2	33.3%	5	83.3%	3	50. %	0	0
	1232	729	59.2%	312	25.3%	151	12.3%	49	4. %	31	2.5%

The variability in the antibody content of adult immune serum as well as adult immune whole blood, and the fact that large amounts must be injected if satisfactory therapeutic results are to be obtained, render the application of these two substances undesirable. Experience has shown that the demand for convalescent serum usually is greater than the available supply and this unfortunate condition has made the application of the method impracticable as a public health measure. This is particularly true during an epidemic when such large quantities are instantly demanded. Since it has been shown that placental extract possesses sufficient antibody content either to protect or to modify measles, and since the percentage of complete failures with this substance compares favorably with the failures encountered when other methods are employed, and in view of the fact that an adequate supply can be maintained, it would appear that the administration of immune globulins from human placentae may be the procedure of choice in the control of measles by public health measures.

REFERENCES

1. Karelitz, S., and Schick, B.: Epidemiologic factors in measles prophylaxis. J. A. M. A. 104:991, 1935.
2. Morales, E. G., and Mandry, O. C.: Relative prophylactic value of convalescent and immune adult measles serum. Am. J. Dis. Child. 39:1214, 1930.
3. Park, W. H., and Freeman, R. G., Jr.: Prophylactic use of measles convalescent serum. J. A. M. A. 87:556, 1936.
4. Levinson, S. O.; McDougall, D., and Thalhimer, W.: Use of convalescent serum for prevention and attenuation of measles. Illinois M. J. 63:258, 1933.
5. Nabarro, D. N., and Signy, A. G.: Convalescent serum in prophylaxis of measles. Brit. M. J. 1:12, 1931. Prevention and control of measles, scarlet fever, and diphtheria in institutions and the home. Brit. M. J. 2:599, 1931.
6. McKhann, C. F.: Ann. Int. Med. In press.

CHAIRMAN GARLAND: I think we will reserve discussion of these papers until they have all been delivered.

The next paper on the program concerns the control of pertussis, by Dr. Francis C. McDonald, of the Boston Floating Hospital.

WHOOPING COUGH*

With Particular Reference to Prophylaxis and Treatment With Vaccines

BY FRANCIS C. MC DONALD, M.D.†

AT a similar meeting held also on a Wednesday morning in the first week of June, nineteen years ago, Dr. John Lovett Morse spoke on "Whooping Cough: The Measures to be Taken for its Control and Prevention." In the nineteen years since that time there has been some improvement in that both the medical profession and laity now seem to realize the seriousness of the disease and do not consider it a "trifling affair" which latter attitude apparently greatly disturbed Dr. Morse[1].

There has been a steady decline in the death rates of the four major communicable diseases of childhood, but this has been disproportionately less for whooping cough and measles as

*From the Boston Floating Hospital and the Department of Pediatrics, Tufts College Medical School.
Read at the Annual Meeting of the Massachusetts Medical Society, Section of Pediatrics, June 5, 1935.

†McDonald, Francis C.—Assistant to the Physician-in-Chief, Boston Floating Hospital. For record and address of author see "This Week's Issue," page 240.

compared with scarlet fever and diphtheria and has resulted in whooping cough now assuming a greater relative importance as a factor in deaths from these infectious diseases. Between 1901 and 1930, in six countries whooping cough was responsible for a third or more of the deaths from the four infectious diseases mentioned. About ten thousand children under fifteen years of age die each year from whooping cough in this country. Roughly, six thousand die in the first year of life, three thousand from the first to the fifth year and one thousand from the fifth to the fifteenth year[2].

When we consider seriously the use of a specific bacterial vaccine for increasing the resistance of the host against a specific disease process, we should feel fairly certain that the microörganism from which the vaccine is made is the cause of that disease. It is necessary then in a discussion of this type for us to review briefly certain labors on the etiology of whooping cough, particularly those relative to the major controversies.

Bordet in 1900, described tiny ovoid gram negative micrococci which were constant in their appearance and showed bipolar staining by toludine phenol blue, and were recovered from the sputum of patients suffering from whooping cough. When other workers agreed with these observations, but on culture recovered influenza-like bacilli which were dissimilar in many ways from the organisms seen on direct observation, he was stimulated to further investigation on the same subject. In 1906, together with Gengou, he published a paper entitled "Le Microbe de la Coqueluche" in which they described the organism before and after culture, and suggested that the organism changed somewhat on prolonged growth. They also noted in guinea pigs the low general toxicity for a bacterial emulsion in contrast to its definite local necrotizing irritation reaction. It is also interesting to note that Bordet in this article commented upon the fact that studies of patients having this disease were less apt to be clouded by undesirable variables if observed in their own homes rather than in hospital units. In later papers he emphasized again that the organism changed its antigenic properties when grown for a long time on artificial media[3, 4].

In 1912, Mallory and Hornor described gram negative bacilli between and upon the cilia and bronchi in fatal pertussis. While attempting to prove the relationship of B pertussis by further animal inoculations, it was found that in animals Bacillus bronchisepticus, considered by some to be the causative organism of distemper, may be easily confused with B pertussis. (There have been rare cases of bronchisepticus infections reported in human beings with symptoms that resemble pertussis.) These two organisms differ only in that the B bronchisepticus is motile and grows more rapidly on some media. They even have some agglutination phenomena in common. The antiserum of B bronchisepticus will agglutinate both B bronchisepticus and old strains of B pertussis. On the other hand B pertussis antiserum will only agglutinate pertussis bacillus[5, 6, 7, 8, 9, 10, 11, 12].

Such discoveries called for a revaluation of all animal transmission experiments in which the disease distemper may have confused the results.

Workers again became less certain of the completeness of their knowledge of the cause of whooping cough when a series of studies on filtrable viruses indicated that some disease processes that were formerly considered to be caused by organisms of the same general family as B pertussis were apparently caused by the concerted action of a filtrable virus and influenza-like organisms. This is most clearly illustrated by Shope's work on Swine Influenza. Also studies on distemper, particularly those by Laidlaw and Dunkin, now seem to indicate that this is primarily a virus disease and that other organisms, including B bronchisepticus, are secondary invaders. These observations were applied, mainly by McCordock, to diseases giving similar pathological pictures in man. Whooping cough, measles and epidemic influenza are frequently complicated by a type of interstitial pneumonia that can be reproduced in animals by injecting vaccine virus together with a suspension of various types of bacteria. This together with the finding of inclusion bodies in the tissues of fatal cases of whooping cough, in a relatively higher percentage than in similar control series, has led McCordock and others to suspect that whooping cough, especially when pneumonia is a complication, may be the result of a virus and B pertussis complex[13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23].

An experiment made on four human beings by MacDonald and MacDonald is surprisingly simple, direct and convincing. A filtrate of a young culture of B pertussis was instilled into the nasopharynx of all four human beings (two of whom had previously been given prophylactic inoculations of vaccine by Sauer's method) and they were quarantined for eighteen days without developing any unusual symptoms. Then a suspension containing about one hundred and forty B pertussis organisms was instilled in each nasopharynx in the same manner as above. The two individuals who had not previously had vaccine prophylaxis developed definite symptoms and clinical, bacteriological and immunological signs of whooping cough. While this experiment leaves us with a feeling that whooping cough is not caused by the filtrate alone and that B pertussis is the cause of the disease, contending investigators claim that it does not completely disprove the thesis that it is caused by a combination of the virus and the Bordet-Gengou bacillus. To settle this uncertainty which arises from the belief that re-

cently cultured organisms may carry the virus or that the virus may still have been present in the nasopharynx after the allowed eighteen day interval (see above), Shibley subcultured B pertussis organisms in their active phase over sixty times during several months. The virulent, frequently subcultured organisms produced symptoms and signs of whooping cough in the experimental animal. The author believes that frequently subculturing effectively separated these organisms from any virus that may have been present in the original culture[24, 25].

In summarizing the known, direct evidence, the following three facts were emphasized by Miller in 1933:

1. The close association of the Bordet-Gengou bacillus with cases of whooping cough.
2. The absence of the organism in the cough droplets of healthy individuals who are not in contact with cases of pertussis.
3. The great frequency with which the organism is isolated in the catarrhal stage, the time observed to be most contagious, and the subsequent diminishing frequency which parallels the observed degree of communicability[26].

MacDonald and MacDonald's human transmission experiments and Rich's, Sauer's and Shibley's animal transmission experiments would make a potent addition to Miller's triad.

We may conclude from this brief survey of the available evidence as concerning the etiology of whooping cough that there are no strong indications that the Bordet-Gengou bacilli are not the cause of the disease. The burden of proof lies with those claiming that a filtrable virus is responsible.

Nicolle and Conor in 1913, are usually given credit for first using a vaccine made from Bordet-Gengou bacilli on a fair-sized group of patients. They used it in a small epidemic in Tunis. Their vaccine was made from organisms recovered from the early cases for treatment of later cases in the same epidemic. In this country Graham of Philadelphia, and Maynard Ladd of Boston, reported their experiences with pertussis vaccine on a relatively few cases in 1911 and 1912. From this time on the reports were numerous and conflicting. Jones in 1928, and Lawson in 1932, submitted theses to the Harvard School of Public Health on whooping cough which carefully appraised the available evidence. Their work was carried on under the supervision of a Whooping Cough Committee appointed in 1925, consisting of Drs. George H. Bigelow, Francis X. Mahoney, Hans Zinsser, Kenneth D. Blackfan, Lawrence Smith and Richard Smith. General conclusions based on this Committee's appraisal of vaccine therapy taken largely from a series of patients studied in The Boston Floating Hospital together with an appraisal of the literature, especially that published by members of the New York City Department of Health, are as follows[27, 28, 29, 30, 31, 32, 33, 34, 35]:

1. The homemade vaccines made from several recently acquired fresh cultures seemed to have been more effective than most stock vaccines.
2. Therapeutic results, if any, were obtained when the vaccine was given early in the disease. Most observers agreed that after the patient had been coughing a week or more vaccines were of little avail.
3. Larger bacterial dosages were thought advisable.
4. It was agreed that heat killed vaccines were not so effective as chemically killed ones.
5. Prophylactic administration appeared more rational and seemed more effective than did therapeutic. However, no controlled prophylaxis by vaccine, allowing a reasonable time interval before exposure, was attempted until Madsen's and Sauer's experience.
6. Most all experienced workers recognized and described the difficulty of making a fair and just estimate of the therapeutic effect. In addition to the variable factors in the disease itself they recognized the unreliability of histories as given by parents. Luttinger illustrated this with the following remarks: "A case free from attacks all day long, having few paroxysms of coughing at night loud enough to awaken the father may be described as a severe case, whereas, another child with frequent severe paroxysms during the day who sleeps through the night leaves the father more indifferent. Young mothers may describe an example of coughing as unusually severe and disturbing. The same attack is trifling to the experienced matron." Histories often must be relied upon for clinical appraisal as not infrequently a really ill child may spend some time in a clinic without demonstrating the symptoms observed in the home.

Von Sholley, Blum and Smith, also of the New York City Health Department, questioned the psychological element in therapy and found that nonspecific influenza vaccine and milk-colored water were as effective as the pertussis vaccines they had been using. This same group of workers pointed out that 24.8 per cent of seven hundred non-vaccinated children who were exposed in their homes escaped the disease.

The fact that some experienced clinicians were favorably impressed by definitely good results from some vaccines and disappointing results from others together with a general realization in research laboratories that all cultures of pertussis bacteria did not behave alike prompted a series of investigations (Krumwiede, Leslie and Gardner) which reaffirmed Bordet's orig-

inal observation that the bacillus changed its antigenic powers when cultured and recultured outside of its host particularly if grown on media free from hemoglobin[36, 37, 38, 39].

This together with the favorable observations of clinical groups who were using vaccines on a large scale made from several recently isolated strains grown on hemoglobin enriched media (mainly in Denmark), revived the waning interest in the use of pertussis vaccines. Madsen's convincing Cutter Lecture given in Boston in 1926 describing the Danish experiences was largely responsible for renewed activity in this country[40, 41].

At the present two vaccines are receiving the greatest attention. They are the vaccine introduced by Krueger and the vaccine introduced by Sauer.

Krueger was impressed by the work of several men which seemed to indicate the importance of the antigenic properties of intracellular constituents of bacteria and emphasized the danger of denaturization of antigenic factors by heat and chemicals. (Sauer's vaccine is killed by .5 per cent phenol.) Krueger places a bacterial suspension previously grown on media enriched with human blood in a mechanical grinder for ten to twelve hours. The ground material is taken up in buffered saline and this suspension is passed through an acetic collodion membrane which is supposed to permit the passage of a "finely dispersed phase" of the organisms and exclude "cell metabolites". A water-clear filtrate is obtained, whereas, the original material was an opalescent suspension[42, 43].

This method has raised the following questions in the minds of some bacteriologists[44].

If the filtrate is water-clear, is one certain that "cell metabolites" are the only masses filtered out? And after the use of these additional procedures how can one be certain of the dosage?

The clinical results following the use of this vaccine have been reported by Frawley, Stallings and Nichols, Munns and Aldrich. Their studies have been largely confined to current epidemics. Prophylactic treatment when given was within the time range of the epidemic and was not effective. Diagnoses were checked by accepted laboratory methods but the results of therapy and prophylaxis are recorded on the basis of subjective symptoms reported by parents. The results as given indicate that the disease was definitely milder and less severe in those cases receiving either prophylactic or therapeutic treatment before the end of the first week. The symptoms were relatively more severe in direct proportion to the duration of the disease before the administration of the vaccine. Reports of this sort are subject to the same difficulties of precise appraisal as were most of those between 1912 and 1928[45, 46, 47, 48].

The vaccine that has excited the most interest in this country recently is that being used by Sauer. His experience in clinical and experimental pertussis extends back to 1918. He used the stock vaccines for ten years with about the same uncertain results that most of the practitioners in this community obtained. He then used a freshly prepared one of his own on one hundred cases after the onset of the disease with unsatisfactory results. From 1928 on, he apparently attacked the problem with a vengeance and seems to have humbly accepted and tried most all of the important suggestions of previous workers even to giving the study of children in their home environment a large place in his series[49, 50, 51, 52, 53, 54, 55, 56, 57, 58].

His knowledge of the parents, the past histories, and the environment of the greater portion of his patients was detailed and personal as they were members of families in his own private practice in his own community. If the greater part of the study were concerned with history and symptoms as given by the parents, this knowledge used properly would tend to minimize the personal equation so far as the parents were concerned. However, a study of prophylaxis resolves itself into determining whether or not the child or the child's contacts have whooping cough rather than an evaluation of the severity of the disease.

The organism from which the vaccine is made grows best on a potato-glycerine-hemoglobin media, only slightly different from that first described by Bordet. Realizing that pertussis is largely a disease of man, and recognizing the tendency of the organism to change its antigenic properties on subculture on other than a "hemoglobin" media, Sauer uses defibrinated human blood instead of defibrinated animal blood. This enables him to dispense with additional, centrifuging and washing measures which would be necessary to remove excess foreign blood protein after the bacterial harvest had been scraped off the media made with foreign blood. He thinks this also lessens the chance of immediate systemic or cutaneous reactions or of sensitization to the animal protein. The human blood is obtained from delivery rooms by collecting cord blood which is ordinarily discarded. The organisms are diluted with normal saline and killed by .5 per cent phenol.

Sauer's vaccine is diluted until 1 cc. contains approximately ten billion organisms. A total of eight cubic centimeters is generally given to each patient making a total dose of eighty billion bacteria as compared with the twenty to twenty-five billion given by Madsen in Denmark and to the seven hundred million to four billion range during the 1912 and 1928 period in this country. At the present time Sauer gives the 8 cc. by three weekly subcutaneous injections (he avoids injecting in pre-

viously injected areas); 1 cc., 1.5 cc. and 1.5 cc. are given into each arm each respective week. The series is given during the non-epidemic period and completed at least four months before expected exposure.

His main results four months to seven years after injection as reported in an address to the New England Pediatric Society this spring were as follows:

One thousand two hundred and nine individuals were vaccinated, with sixty-two definite exposures and 152 casual exposures with six resulting cases of whooping cough. Three of the six contracted the disease shortly after recovery from measles. (This does not include an institution series of 400.)

Sauer, himself, has emphasized the need of further studies over a relatively longer period of time. As the majority of the group mentioned above have been inoculated since January of 1933, we are particularly interested in the subsequent history of his first group of 394 patients first reported upon in January 1933, which group had been inoculated during the five preceding years. Of the 394 individuals twenty-seven were from families having one or more other children, thirty-one of whom were left uninoculated as controls. All thirty-one of these controls contracted pertussis while all except one of their inoculated brothers and sisters lived with them throughout the incubation, catarrhal and paroxysmal stages without contracting the disease. Of the remainder of the original 394, 124 were reported to have been definitely exposed to the disease, yet none contracted it.

The Council on Pharmacy and Chemistry of the American Medical Association reconsidered vaccines of B pertussis (Sauer's) in March 1934, after having previously omitted them from New and Nonofficial Remedies in 1931. They granted that Sauer's work appeared promising, but before rerecognition of the value of pertussis vaccine expressed a desire that a larger series of cases be collected and that they be gathered from a less localized geographical area[59].

The Michigan Board of Health and the commercial laboratories are now manufacturing vaccines following Sauer's method. However, in making them on a large scale they find it difficult to collect human blood in sufficient quantities for use in the potato-glycerine media. Hence, animal blood is being used in the preparation of these vaccines. Kendrick, representing the Michigan State Board of Health, sees no reason why animal blood should not be used. Sauer still feels that until the value of the vaccine has been proved more definitely the technique of manufacture should not be altered. At the present time he does not use his vaccine for other than prophylaxis. This is in agreement with past clinical experience and with the general feeling of many bacteriologists that the development of immunity as stimulated by vaccines is a matter of several weeks rather than of a few days.

In conclusion, while we cannot ignore the possibility that a filtrable virus may play a part in the disease picture, the proponents of vaccine prophylaxis have offered a program that gives promise of preventing whooping cough in the young patient. A question that naturally arises is: Are there any serious contraindications to the use of the vaccine? Two deaths are reported in Madsen's series in infants vaccinated because there was whooping cough in the immediate family. One was newly born. Four days after an inoculation of .1 cc., a second of .15 cc. was given, thirty minutes later the patient died with hiccoughs, cyanosis and contracture of the extremities. The second fatality occurred in an eight day old premature infant. The first inoculation was .1 cc., the second .2 cc. after a three day interval. Two hours later death occurred suddenly with slight cyanosis.

These deaths, particularly the latter, occurred in an uncertain age period where the vaccine may have played no direct part in the fatalities. However, the use of the vaccine made from organisms grown on media enriched with human blood prepared as advocated by Sauer appears reasonably safe and rational if given after the seventh month of life with the series completed at least four months before exposure. Sauer does not think that immunization should be attempted very soon after recovery from other diseases (N.B. measles) or within several months of other immunization. (Diphtheria, smallpox, scarlet fever.)

The Bordet-Gengou bacillus does play an important rôle in the disease picture of whooping cough. While vaccine therapy appears to be of doubtful value, vaccine prophylaxis as outlined by Sauer offers definite promise. However, several years of further use will be necessary for final evaluation.

REFERENCES

1. Morse, John Lovett: Whooping cough; the measures taken for its control and prevention. Boston M. & S. J. 175:723, 1916.
2. Epidemiological Report Health Commission—League of Nations: Relative importance of the principal infectious diseases of childhood. R. E. 173: (May, June) 1934.
3. Bordet, J., et Gengou, O.: Le microbe de la coqueluche. Ann. de l'Inst. Pasteur 20:731, 1906.
4. Bordet, J., et Sleeswyk: Sérodiagnostic et variabilité des microbes suivant le millieu de culture. Ann. de l'Inst. Pasteur 24:476, 1910.
5. Mallory, F. B., and Hornor, A. A.: Pertussis; the histological lesion in the respiratory tract. J. M. Research 22:115, 1912-13.
6. Mallory, F. B.; Hornor, A. A., and Henderson, F. F.: The relation of the Bordet-Gengou bacillus to the lesion of pertussis. J. M. Research 22:391, 1912-13.
7. Smith, T.: Some bacteriological and environmental factors in the pneumonias of lower animals with special reference to the guinea-pig. J. M. Research 24:291, 1913-14.
8. Mallory, F. B.: The pathological lesion of whooping cough. Boston M. & S. J. 169:378 and 575, 1913.
9. Rhea, Lawrence J.: The comparative pathology of the tracheal and bronchial lesions produced in man by b. pertussis (whooping-cough) and those produced in dogs by b. bronchisepticus (canine distemper). J. M. Research 27:471, 1915.

10. Ferry, N. S., and Noble, A.: Studies relative to the apparent close relationship between bact. pertussis and b. bronchisepticus. J. Bact. 3:193, 1918.
11. Torrey, J. C., and Rahe, A. H.: Studies in canine distemper. J. M. Research 22:291, 1912-13.
12. Brown, J. H.: Bacillus bronchisepticus infection in a child with symptoms of pertussis. Bull. Johns Hopkins Hosp. 38:147 (Feb.) 1926.
13. Duran-Reynals, F.: The effect of extracts of certain organs from normal and immunized animals on the infecting power of vaccine virus. J. Exper. Med. 50:327, 1929.
14. Shope, Richard E.: Swine influenza. J. Exper. Med. 54:349, 361, 373; 1931.
15. Muckenfuss, R. S.; McCordock, H. A., and Harter, S. J.: A study of vaccine virus pneumonia in rabbits. Am. J. Path. 7:552, 1931. Ibid. 8:63, 1932.
16. McCordock, H. A., and Muckenfuss, R. S.: The similarity of virus pneumonia in animals to epidemic influenza and interstitial bronchopneumonia in man. Am. J. Path. 9:221, 1933.
17. McCallum, W. G.: Pathology. J. A. M. A. 70:1153 (Apr.) 1918.
18. McCordock, H. A., and Smith, Margaret G.: Intranuclear inclusions; incidence and possible significance in whooping cough and in a variety of other conditions. Am. J. Dis. Child. 47:771, 1934.
19. McCordock, H. A.: Intranuclear inclusions in pertussis. Proc. Soc. Exper. Biol. & Med. 29:1288 (June) 1932.
20. Rich, A. R.: On etiology and pathogenesis of whooping cough. Bull. Johns Hopkins Hosp. 51:346 (Dec.) 1932.
21. Rich, A. R.; Long, P. H.; Brown, J. H.; Bliss, E. A., and Holt, L. E., Jr.: Experiments upon cause of whooping cough. Science 76:330 (Oct. 7) 1932.
22. Laidlaw, P. P., and Dunkin, G. W.: Studies in dog-distemper. J. Comp. Path. & Therap. 39:201, 213, 222; 1926; 41:1 (Mar. 31) 1927; 44:1 (Mar. 31) 1931.
23. Green, R. G.: Distemper in the silver fox. Proc. Soc. Exper. Biol. & Med. 23:677, 1925-26.
24. MacDonald, H., and MacDonald, E. J.: Experimental pertussis. J. Infect. Dis. 53:328 (Nov.-Dec.) 1933.
25. Shibley, G. S.: Etiology of whooping cough. Proc. Soc. Exper. Biol. & Med. 31:576 (Feb.) 1934.
26. Miller, J. J.: Etiology of whooping cough. J. A. M. A. 100:681 (Mar. 4) 1933.
27. Nicolle, C., et Conor, A.: Vaccinotherapie dans la coqueluche. Compt. Rend. Acad. d. sc. Paris 46:1849, 1913.
28. Graham, E. E.: The treatment of pertussis with vaccine. Am. J. Dis. Child. 3:41 (Jan.) 1912.
29. Ladd, M.: Vaccines in the treatment of pertussis. Arch. Pediat. 24:581 (Mar.) 1912.
30. Pierret, R.: Present situation in France of biological prophylaxis and treatment in certain contagious diseases, especially whooping cough and measles. Proc. Roy. Soc. Med. 25:1329, 1932.
31. Jones, John L.: A study of certain aspects of whooping cough. Thesis submitted to the faculty of the Harvard School of Public Health April 15, 1928.
32. Lawson, George M.: Studies on bacillus pertussis. Thesis submitted to the faculty of the Harvard School of Public Health July, 1932.
33. Luttinger, Paul: Whooping-Cough; its treatment and prophylaxis based on the Bordet-Gengou etiology. N. Y. Med. J. 101:1043 (May 22) 1915.
34. Von Sholley, A. I.; Blum, J., and Smith, L.: Therapeutic value of pertussis vaccine in whooping cough. J. A. M. A. 68:1451 (May 19) 1917.
35. Hess, A. F.: The use of a series of vaccines in the prophylaxis and treatment of an epidemic of pertussis. J. A. M. A. 63:1007 (Sept. 19) 1914.
36. Povitzky, O. R.: Improved methods for isolation and later cultivation of b. pertussis. J. Infect. Dis. 32:8 (Jan.) 1923.
37. Mishulow, L.; Oldenbusch, C., and Scholl, M.: Potency of stored pertussis vaccines. J. Infect. Dis. 41:169 (Aug.) 1927.
38. Krumwiede, C.; Mishulow, L., and Oldenbusch, C.: Existence of more than one immunologic type of b. pertussis. J. Infect. Dis. 32:22, 1923.
39. Leslie, P. H., and Gardner, A. D.: Phases of haemophilus pertussis. J. Hyg. 31:423, 1931.
40. Madsen, T.: Whooping cough; its bacteriology, diagnosis, prevention and treatment. Boston M. & S. J. 192:50 (Jan. 8) 1925.
41. Madsen, T.: Vaccination against whooping cough. J. A. M. A. 101:187 (July 15) 1933.
42. Krueger, A. P.: Method for preparation of bacterial antigens. J. Infect. Dis. 53:237, 1933.
43. Krueger, A. P.; Nichols, V. C., and Frawley, J. M.: Preparation of active undenatured antigen from hemophilus pertussis. Proc. Soc. Exper. Biol. & Med. 30:1097 (May) 1933.
44. Fothergill, LeRoy: Personal Communication.
45. Frawley, J. M.: Immunization of school children against whooping cough. J. Pediat. 4:184 (Feb.) 1934.
46. Frawley, J. M.; Stallings, M., and Nichols, V. C.: New pertussis antigen; preliminary clinical report. J. Pediat. 4:179 (Feb.) 1934.
47. Munns, G. F., and Aldrich, C. A.: Hemophilus pertussis endoantigen (Krueger) uses in 122 cases. J. Pediat. 5:590 (Nov.) 1934.
48. Stallings, M., and Nichols, V. C.: Treatment of whooping cough with active undenatured antigen. Am. J. Dis. Child. 48:1183 (Dec.) 1934.
49. Sauer, L. W., and Hambrecht, L.: Whooping cough; vaccine therapy or early diagnosis. J. A. M. A. 91:1861, 1928.
50. Sauer, L. W., and Hambrecht, L.: Experimental whooping cough. Am. J. Dis. Child. 37:732 (Apr.) 1929.
51. Sauer, L. W., and Hambrecht, L.: Whooping cough; site of the lesion. Arch. Path. 8:944 (Dec.) 1929.
52. Sauer, L. W., and Hambrecht, L.: Whooping cough; early diagnosis by the cough plate method. J. A. M. A. 95:263 (July 26) 1930.
53. Sauer, L. W.: Whooping cough; résumé of a 7 years' study. J. Pediat. 2:740 (June) 1933.
54. Sauer, L. W.: Whooping cough; study in immunization. J. A. M. A. 100:239 (Jan. 28) 1933.
55. Sauer, L. W.: Immunization with bacillus pertussis vaccine. J. A. M. A. 101:1449 (Nov. 4) 1933.
56. Sauer, L. W.: Preparation of bacillus pertussis vaccine for immunization. J. A. M. A. 102:1471 (May 5) 1934.
57. Sauer, L. W.: Immunization against whooping cough. Am. J. Dis. Child. 49:69 (Jan.) 1935.
58. Sauer, L. W.: Whooping cough and its prevention. Read at the New England Pediatric Society March 22, 1935.
59. Preliminary report of the Council on Pharmacy and Chemistry: Pertussis vaccine. J. A. M. A. 102:692 (Mar. 3) 1934.
60. Kendrick, P., and Edlering, G.: Significance of bacteriological methods in diagnosis and control of whooping cough. Am. J. Pub. Health 25:147 (Feb.) 1935.

CHAIRMAN GARLAND: The third paper on the program will be on scarlet fever, by Dr. Gaylord W. Anderson, Director of the Division of Communicable Diseases of the State Department of Public Health.

THE PRESENT STATUS OF SCARLET FEVER PREVENTION*

BY GAYLORD W. ANDERSON, M.D.†

TWELVE years have now elapsed since the Dicks announced their basic scarlet fever studies showing the etiological relationship of certain hemolytic streptococci[1, 2] the production of a toxin injection of which might be used in small quantities to determine susceptibility[3] and in large doses to enhance the human resistance[4, 5] and the production of a specific antitoxin which might be used for the treatment of the disease[6]. The ensuing years have seen a large number of subsequent studies and the presentation of much material the exact interpretation of which is far from clear, yet so far as I am aware, none of it has upset any of the fundamental facts originally established.

In attempting at this time to bring before you a brief critical analysis of the present status of scarlet fever prevention, it seems best that the conclusions should be based solely upon the findings of other observers than the Dicks and their immediate associates[7, 23]. My decision to do so was based primarily on the thought that the true practical value of such methods lies not in the results claimed by those originators who may have acquired special skill in a certain technique and who because of a pardonable pride in achievement may unwittingly view all doubtful data in their best possible light, but

*From the Massachusetts Department of Public Health.
Read before the Section of Pediatrics at the One Hundred and Fifty-Fourth Annual Meeting of the Massachusetts Medical Society, Boston, June 5, 1935, as part of a symposium on the control of certain communicable diseases.

†Anderson, Gaylord W.—Deputy Commissioner and Director of the Division of Communicable Diseases, Massachusetts Department of Public Health. For record and address of author see "This Week's Issue," page 240.

rather in the results obtained by others less familiar with the technique and approaching the problem without the least element of possible prejudice. They who come to criticize but remain to applaud are the best supporters of any cause. I have further thought it better to omit the results claimed by the Dicks because unfortunately many of the articles are so lacking in the details essential for a critical analysis as to leave doubt as to the exact value of the figures. This doubt is enhanced by claims which, if substantiated, would place the Dick test and the immunizing toxin in a separate class by itself, far superior in reliability and value to any other known biologic agents, a rating that cannot be accorded to them by the findings of other observers no matter how favorable their results. It has, therefore, appeared wise to base our opinions solely on the work of investigators other than those associated with the Scarlet Fever Committee.

THE DICK TEST

The first problem that must be considered is the reliability of the Dick test as an index of immunity to scarlet fever. To what extent can it be said that a person with a negative Dick test is immune to the disease? Studies of hospital nurses and internes, the majority of whom are at one time or another exposed to scarlet fever, furnish probably the most severe test. Thus Knights[24] on the basis of a one year's experience with a small group in Providence, reports an attack rate of 2.1 per cent among the Dick negative nurses as contrasted with a rate of 18 per cent for the Dick positive. Peacock, Werner and Colwell[25] in Chicago report only one questionable case of scarlet fever among 186 nurses with a negative test, and two out of five whose test was positive. Faulds[26], at the Royal Infirmary in Glasgow, found no cases among 298 Dick negative nurses for a three year period. Boynton[27], studying the nurses in the University of Minnesota School of Nursing, found one case among 376 negative reactors, whereas seven of 619 that were neither tested nor immunized contracted the disease. Toyoda[28] and his associates, working in Japan, found no cases among 157 immune hospital attendants and ten cases in forty-three susceptibles. In Edinburgh, Benson and Rankin[29] report but five cases among 1,063 negative reactors intimately exposed to the disease, and fifty-two cases among 363 Dick positives who were in general less intimately exposed. In a survey of the hospitals in Massachusetts, Anderson and Reinhardt[30] found for a period ranging from one to eight years only nine cases of scarlet fever in 1,337 nurses with a negative reaction, whereas thirty out of 234 (12.8 per cent) of those who had a positive test and were not immunized contracted the disease.

These results from a group whose exposure to scarlet fever has been unusually intimate indicate a high degree of specificity for the Dick test. The results are substantiated by institutional and community studies. Probably the largest series is that of Sparrow[31] who tested 23,657 children in Warsaw during 1925 to 1927, of whom forty-five per cent, were Dick negative. During the ensuing winter, when scarlet fever was at a high level, there were but four cases

TABLE 1

RELIABILITY OF DICK TEST AMONG NURSES

Observer	Dick Neg.	Cases of S. F.	Rate	Dick Pos.	Cases of S. F.	Rate
Knights	143	3	2.1	22	4	18%
Peacock, etc.	186	1	0.5	5	2	40%
Faulds	298	0	0	—	—	—
Boynton	376	1	0.3	—	—	—
Toyoda et al.	157	0	0	43	10	23%
Benson and Rankin	1063	5	0.5	363	52	14%
Anderson and Reinhardt	1337	9	0.7	234	30	13%
	3560	19	0.5	667	98	15%

in this immune group as contrasted with 127 in the susceptible group. Nesbit[32] has found comparable data in the school system of Gary, Indiana, one year's observation showing no cases of scarlet fever in a group of approximately 1,000 spontaneously immune children. Albesco[33] reported an attack rate of 0.2 per cent among 3,051 Dick negative Roumanian children, as compared with 4.6 per cent among an unimmunized Dick positive group. Kiefer[34], Molitch[35] and others have reported comparable results in institutions. In Japan, however, Toyoda and his associates[28] found forty-eight cases among 7,186 Dick negative school children, an attack rate of 0.7 per cent, as contrasted with 209 cases (4.4 per cent) in a group of 4,733 susceptibles. They[36] quote the experience of Ozaki, likewise in Japan, who found an attack rate of 10.6 in a small susceptible group as contrasted with a rate of 0.1 in a Dick negative group of 1495 children. Similarly, in a group of home contacts, Toyoda[28] reported no cases among seventy-three immunes and six, or 11.5 per cent, among fifty-two susceptibles. More recently Gordon and his associates[37], working in Detroit, found in a study of family contacts an attack rate of two per cent among 723 Dick negatives, as contrasted with thirteen per cent among 566 susceptibles.

These results have been selected as typical examples of the findings of investigators in many different places. While they differ slightly in the exact degree of immunity indicated by a negative Dick test, they all agree, however, in indicating that the Dick test is in practice an extremely sensitive measure of resistance to clinically recognizable scarlet fever. It is true that

failures of the test will be encountered, but in this respect it differs in no regard from the Schick test. As a measure of human resistance to the disease it is therefore an extremely valuable and reasonably reliable addition to our control procedures.

TABLE 2

RELIABILITY OF DICK TEST

COMMUNITY STUDIES

	Dick Neg.	Cases of S. F.	Rate	Dick Pos.	Cases of S. F.	Rate
Sparrow	10,600*	4	0.04%*	13,000*	127	1.0%*
Nesbit	1,080	0	0	—	—	—
Albesco	3,051	7	0.2	329	15	4.6
Toyoda	7,186	48	0.7	4,733	209	4.4
Ozaki	1,495	2	0.1	47	5*	10.6

*Figure calculated from author's data.

TABLE 3

RELIABILITY OF DICK TEST

FAMILY CONTACTS

	Dick Neg.	Cases of S. F.	Rate	Dick Pos.	Cases of S. F.	Rate
Toyoda	63	0	0%	52	6	11.5%
Gordon	723	15	2	566	73	13

PASSIVE IMMUNIZATION

Passive immunization is possible through the use of three different products. Antitoxin, originally recommended by the Dicks, will unquestionably give a protection lasting from two to four weeks. It has, however, fallen into more or less disrepute because of the subsequent serum reactions which are frequently more severe than the prevailing type of scarlet fever. There is, however, no doubt as to its efficacy. Blood serum from those convalescent from the disease may likewise be used where obtainable. Here also there is little doubt as to its efficacy if used in sufficient quantities, though the optimum dosage for different patients has not been determined. Meader[38] reported an eighty per cent reduction of secondary cases through a dosage of 7.5 cc., though Gordon[39] finds larger doses preferable. The principal problem here, however, is that of obtaining enough serum for the needs of a community. More recently McKhann[40] has demonstrated a similar protective action from an extract of the placenta. All of these methods confer, however, merely a temporary protection and are of no lasting value.

ACTIVE IMMUNIZATION

Evaluation of the results obtained through active immunization using graduated doses of the Dick toxin is extremely difficult because of variations encountered in the dosages used. The early work of the Dicks and others must be largely disregarded because the dosages used were so small as compared with the present-day schedule that the results are not comparable. Yet in spite of the improvements coming from larger doses, one cannot help wondering if the advantage of improved level of protection may not have been outweighed by the obvious disadvantages of reactions and number of injections.

The protection obtained from these injections can be measured either in terms of their effect on the Dick test or the protection conferred to subsequent exposure to the disease. It can be generally stated on the experience of many different observers that the present-day dosages of 500, 2,000, 8,000, 25,000 and 80,000 skin test doses (S.T.D.) will cause a positive Dick test to become negative in about 90-95 per cent of the persons so treated. With smaller doses the percentage will fall off, though by no means proportionately to the decrease in units.

The clinical evidence as to the immunity so established is best measured in the groups of hospital attendants. In table 4 are summarized many of the data now available. Although there was some variation in the doses of toxin used, the total number of units rarely fell below a total of 80,000. The difference between the low attack rate of 0.5 for the immunized nurses and fourteen per cent for the known susceptibles permits of no other interpretation than that the toxin injections have in reality conferred a high level of protection.

TABLE 4

TOXIN IMMUNIZATION

SCARLET FEVER ATTACK RATE AMONG NURSES

Observer	Immunized	Cases	Rate	Dick Pos.	Cases	Rate	No Test	Cases	Rate
Knights[24]	26	0	0	22	4	18%	78	7	10.0%
Peacock et al.[25]	38	0	0	5	2	40%	—	—	—
Toyoda[36]	—	—	—	43	10	23%	—	—	—
Boynton[27]	164	0	0	—	—	—	619	7	1.1%
Platou[41]	149*	0	0	—	—	—	861	55	6.4
Benson and Rankin[20]	—	—	—	363	52	14%	—	—	—
Anderson and Reinhardt[30]	983	7	0.7%	234	30	13%	3737	118	3.1
	1360	7	0.5%	667	98	15%	5295	187	3.5%

*Includes some children as well as nurses.

Similar data are available for community studies. Thus Nesbit and Thompson[42] report that in the Gary schools there have been approximately 10,000 given the toxin and during eight years only nine cases of scarlet fever among those who were negative on the retest. Sparrow[31], using dosages of but 3,500 S.T.D. found a reduction to one-fourth of that in the non-immunized group. Albesco[33] in Roumania, using small doses, never exceeding a total of 13,500 S.T.D. of toxin, found in two years an attack rate of 1.2 per cent among the immunized as compared with 4.6 per cent among a much smaller group of non-immunized susceptible children. Toyoda and his associates[36], using dosages of 25,000 to 40,000 S.T.D., found an attack rate in Dairen of but 0.1 per cent among the immunized as contrasted with 6.2 per cent among the non-immunized. They also quote the experience of Ozaki, who found a rate of 0.3 per cent among the immunized and 2.4 per cent among the non-immunized. To these series might be added the experience of numerous institutions in this country and abroad showing that in the face of an outbreak those immunized with the Dick toxin were protected, although new admissions that were not so protected contracted the disease. It is therefore a fair statement to say that immunization with the Dick toxin in adequate doses confers to a person previously susceptible a level of resistance against clinically recognizable scarlet fever that is comparable to that shown by a negative Dick test, and furthermore, that this protection is probably comparable in effectiveness to that conferred against diphtheria through the use of the commonly accepted immunizing agents.

DURATION OF IMMUNITY

The exact measure of the duration of immunity is hard to determine because it usually is impossible to measure the extent to which the rapidly developed immunity produced by the toxin may have been maintained or enhanced by subsequent exposures to new infecting doses of the causative organisms. The results among nurses would suggest an immunity of at least three years though in this group especially there is, theoretically at least, opportunity for much reënforcement of the resistance through frequent exposures to subinfecting doses. Nesbit and Thompson[42] report eighty-one per cent immunity by Dick test among 171 children immunized five or more years previously, while Bull[43] found sixty-four per cent negative and thirty-six per cent slightly positive among fifty immunized eight years previously. There are few published data available as to the permanence of a negative reaction among those found naturally immune. In studies carried on by the Department we have found in an institution where scarlet fever was not prevalent that 644 or eighty-nine per cent of a group of 725 originally negative were still Dick negative four years later, a finding that compares quite favorably with similar studies of the permanence of a negative Schick test in a similar institution under similar conditions. On the other hand, of forty-one susceptible children left unimmunized in the same institution, thirty-one or seventy-five per cent were still susceptible at the end of four years.

REACTIONS

Whatever may be the differences of opinion as to the reliability of the Dick test and effectiveness of immunization with Dick toxin, there is a surprising unanimity of opinion as to the severity of the reactions encountered from the toxin injections. These reactions may be briefly described as nausea, vomiting, headache, fever, rarely sore throat and occasionally a typical scarlatiniform rash with or without desquamation. Most observers mention "severe" reactions in as high as ten to fifteen per cent of those so treated. These reactions have been sufficiently severe so that they have unquestionably militated against the ready acceptance of scarlet fever immunization in many instances. Thus many nurses have preferred to take their chances with the present mild form of the disease. In community programs there has been a markedly greater falling off in clinic attendance[44] than is encountered in similar diphtheria immunization programs. Melnick[45], in institution work, was even prompted to recommend a "pre-immunization" preparation of a laxative, diet restricted to fruit juices, cereal, water, tea and toast on the day of the injection, restricted activities, and injection given at night and just before a week-end so as to avoid absence from school of those who would be in bed the following day. This latter is probably an extreme point of view, but the fact is inescapable that a definite percentage of those who are so treated will probably require bed care during the ensuing twenty-four to forty-eight hours. Unfortunately, it does not seem possible to avoid these reactions by reducing the dosage, as they have been described following doses that are but a fraction of that recommended by the Scarlet Fever Committee as needed for a lasting immunity. It is necessary, therefore, to turn to other types of preparations in search of immunizing agents that can be given without unpleasant reactions.

"TOXOID" IMMUNIZATION

Since the early days of toxin immunization many attempts have been made to modify the toxin in a manner comparable to that used in the preparation of diphtheria toxoid, in the hope of obtaining a potent immunizing agent that would at the same time be devoid of the

severe toxin reactions. Many preliminary but inconclusive reports have been published in this country and abroad, leaving the question still very much open to debate.

The most recent attempt in this country to produce a scarlet fever toxoid has been that of Veldee, who in 1930[46] and 1931[47] reported preliminary studies to this end, and in 1933[48] reported its use on several hundred children. Some of this work was carried on in collaboration with the Massachusetts Department of Public Health. Beginning with these studies in the fall of 1931 and continuing through the present day, the Department has been pursuing extensive studies of this formalized toxin solution prepared according to the methods of Veldee*. In the course of the studies we have Dick tested over 15,000 children and administered the material to 6,000 to 7,000 children. These studies have been carried on in twenty-seven different institutions, in the nurses' training schools of several hospitals, and more recently on a community-wide basis in ten cities and towns. In some of these institutions the studies have been under way for about four years, and in the majority for over two years. In two of the communities, over a year has now elapsed since the immunization work was completed and we are beginning to obtain results of real value. The work in the other communities has been concluded during the past winter or is at the present moment being finished so that no conclusions will be available for at least another year.

I am not prepared at the present moment to report to you in detail as to the results of these studies, other than in generalities. The material is being given in but three injections three weeks apart. The reactions it produces are no more severe than are encountered in diphtheria immunization and markedly less severe than with the Dick toxin. This is attested to not only by the physicians, superintendents, nurses and teachers who have seen its use, but also by the parents who have brought their children back for subsequent injections equally readily as for diphtheria. As measured by the Dick test the resulting immunity is not so high as obtained through the five doses of toxin, as recommended by the Dicks. Work of the past winter has strongly suggested, however, that after "toxoid" immunization, control tests with a heated toxin are essential to eliminate false positive reactions. These may give to the retests a more flattering appearance. The value of the "toxoid" when measured by the clinical test of protecting against clinically recognizable scarlet fever has been strikingly shown in a virtual absence of the disease from the institutions where it has been used. In the two communities where it has been possible to evaluate results, there has been demonstrated a marked degree of protection for those children given the "toxoid". Clinically, therefore, it is fair to say that at present it appears to offer an immunizing agent that, through its ready acceptance because of mild reactions, may be used so extensively as to lessen materially the incidence of scarlet fever even though the protection conferred in an individual case may be less than with the more powerful Dick toxin.

*Recently the Dicks[49] have questioned the existence of a true toxoid, maintaining that the immunizing effect of the solution is due solely to the residual toxin that has been unaffected by the formalin. Further studies are needed to clarify this situation.

"SCARLATINAL TONSILLITIS"

In the midst of the studies of scarlet fever immunization the suggestion has frequently been advanced that immunization by protecting the individual against the rash would be followed by an increase in sore throats of streptococcal origin, which would in reality be scarlet fever without a rash or, as Benson has named them, "scarlatinal tonsillitis". Kinloch, Smith and Taylor[50], on the basis of a single year's experience, reported an increase in cases of tonsillitis among nurses after immunization has been carried on. Gordon and his associates[37] in a study of family contacts with cases of scarlet fever found that ten per cent of the Dick positives developed tonsillitis without rash whereas but 4.5 per cent of the Dick negatives had a similar experience, a rather surprising difference were the Dick test to indicate merely a resistance to the toxin and not to the infection itself. Benson and Rankin[29] in their studies at the City Fever Hospital of Edinburgh found no increase in tonsillitis among the probationer staff, the preimmunization attack rate 1919-1925 being 22.1 as compared with a rate of 16.5 for the years 1926-1933 after immunization had become a routine. They have further studied the cross infection rate of scarlet fever to diphtheria patients as a test of the hypothesis that immunized nurses might more readily be carriers of the hemolytic streptococci of scarlet fever. Their results showed that during the preimmunization period 3.5 per cent of the diphtheria patients contracted scarlet fever, whereas during the years subsequent to the inauguration of scarlet fever immunization among the nursing staff only 2.1 per cent developed a cross infection of scarlet fever. They conclude that "there is no evidence that clinical scarlet fever has been replaced by 'scarlatinal tonsillitis' in successfully immunized nurses and an immunized nursing staff does not increase the risk of conveyance of scarlet fever to non-scarlatinal patients." I know of no evidence that would prove the incorrectness of this statement.

SUMMARY

In summarizing this analysis of the present status of scarlet fever immunization, I hope I may be pardoned if I express a personal estimate as to the value of the data here presented. Many of us, and I include myself among the group,

have been so impressed with the reactions encountered from Dick toxin immunization that we have hesitated to recommend its use. This would be justifiable were it not for the fact that we have rationalized our hesitancy upon a doubt as to the validity of the Dick test and the efficacy of the toxin. The issues are in reality quite separate, but we have permitted them to become confused in our minds. We must, I believe, recognize that the Dick test is in reality a very reliable index of immunity to scarlet fever, little if any inferior to the Schick test in its own field. We should also acknowledge that with the Dick toxin susceptible persons can actually be protected against scarlet fever with a measure of success little short of that seen in diphtheria. Its only limitations are the reactions encountered which will probably for a long time to come make its use impractical on a community basis in public clinics. In institutions, and in the hands of the private practitioner, on the other hand, who can explain in advance the possibility of reactions and thus avoid alarm on the part of the parent, it offers an extremely effective means of scarlet fever control and is deserving of more extensive use than at present accorded to it.

REFERENCES

1. Dick, G. F., and Dick, G. H.: J. A. M. A. 81:1166, 1923.
2. Dick, G. F., and Dick, G. H.: J. A. M. A. 82:301, 1924.
3. Dick, G. F., and Dick, G. H.: J. A. M. A. 82:265, 1924.
4. Dick, G. F., and Dick, G. H.: J. A. M. A. 82:544, 1924.
5. Dick, G. F., and Dick, G. H.: J. A. M. A. 83:84, 1924.
6. Dick, G. F., and Dick, G. H.: J. A. M. A. 82:1246, 1924.
7. Dick, G. F., and Dick, G. H.: Am. J. Public Health. 14: 1022, 1924.
8. Dick, G. F., and Dick, G. H.: J. A. M. A. 84:1477, 1925.
9. Dick, G. F., and Dick, G. H.: Northwest Med. 25:111, 1926.
10. Dick, G. F.: J. Mich. State Med. Soc. 26:351, 1927.
11. Dick, G. F.: Northwest Med. 27:1, 1928.
12. Dick, G. F.: W. Va. Med. J. 24, 441, 1928.
13. Hektoen, L., and Johnson, C.: J. Prev. Med. 2:289, 1928.
14. Dick, G. F., and Dick, G. H.: Am. J. Dis. Child. 38:905, 1929.
15. Dick, G. H.: Illinois M. J. 57:22, 1930.
16. Dick, G. F.: Internat. Clin. 1:150, 1930.
17. Dick, G. F.: Kentucky M. J. 29:172, 1931.
18. Rhoads, P. S.: J. A. M. A. 97:153, 1931.
19. Rhoads, P. S.: J. Iowa Med. Soc. 21:443, 1931.
20. Dick, G. F.: M. Officer 49:75, 1933.
21. Dick, G. F., and Dick, G. H.: Edinburgh M. J. 41:1, 1934.
22. Hektoen, L., and Johnson, C.: J. A. M. A. 102:41, 1934.
23. Dick, G. H.: Journal-Lancet 54:289, 1934.
24. Knights, E. M.: J. Lab. and Clin. Med. 14:614, 1929.
25. Peacock, S. C.; Werner, M., and Colwell, C.: A. J. D. C. 44:494, 1932.
26. Faulds, J. S.: J. Hyg. 33:353, 1933.
27. Boynton, R. E.: Am. J. Nursing 34:330, 1934; also Minnesota Med. 17:165, 1934.
28. Toyoda, T.; Moriwaki, J., and Futagi, Y.: J. Infect. Dis. 46:186, 1930.
29. Benson, W. T., and Rankin, A. L. K.: Lancet 2:1357, 1934; see also Benson, W. T.: Edinburgh M. J. 35:617, 1928.
30. Anderson, G. W., and Reinhardt, W. I.: J. Inf. Dis. to be published.
31. Sparrow, H.: Presse méd. 36:549, 1928.
32. Nesbit, O. B.: J. A. M. A. 84:805, 1925.
33. Albesco, V.: Compt. rend. Soc. de biol. 115:1326, 1934.
34. Kiefer, G. L.: J. A. M. A. 91:1885, 1928.
35. Molitch, M.: J. A. M. A. 102:2021, 1934.
36. Toyoda, T.; Moriwaki, J.; Futagi, Y., and Okamoto, M.: J. Inf. Dis. 46:219, 1930.
37. Gordon, J. E.; Badger, G. F.; Darling, G. B., and Schooten, S. S.: Am. J. Public Health 25:531, 1935.
38. Meader, F. M.: J. A. M. A. 94:622, 1930.
39. Gordon, J. E.: Monthly Report of Herman Kiefer Hospital, Detroit, June, 1934.
40. McKhann, C. F., and Chu, F. T.: J. Inf. Dis. 52:268, 1933.
41. Platou, E. S.: J. of Ped. 5:531, 1934; see also Minn. Med. 15:697, 1932.
42. Nesbit, O. B., and Thompson, S.: Am. J. Public Health 24:634, 1934.
43. Bull, H. G.: J. A. M. A. 101:363, 1933.
44. Schwartz, A. B.: J. of Ped. 5:282, 1934.
45. Melnick, T.: Arch. Pediat. 50:158, 1933.
46. Veldee, M. V.: Public Health Reports 45:1827, 1930.
47. Veldee, M. V.: Public Health Reports 46:693, 1931.
48. Veldee, M. V.: Public Health Reports 48:549, 1933.
49. Dick, G. F., and Dick, G. H.: J. A. M. A. 103:1362, 1934.
50. Kinloch, J. P.; Smith, J., and Taylor, J. S.: J. Hyg. 26:327, 1927.

CHAIRMAN GARLAND: The next paper on the program is on diphtheria, by Dr. Elliott S. Robinson, Director of the State Antitoxin and Vaccine Laboratory.

THE CONTROL OF DIPHTHERIA*

BY ELLIOTT S. ROBINSON, M.D.†

THE experience of the last twenty years, more particularly the last ten, has shown clearly that the most satisfactory method of controlling diphtheria consists in active immunization of as many children as possible. There is every reason to believe that the more thoroughly and completely this practice is carried out, the lower will be the incidence of the disease in the community. Therefore, the discussion of the control of this disease consists not in considering whether active immunization should or should not be carried out but upon what immunizing agent should be used for that purpose.

The first immunizing agent to be used on a large scale was diphtheria toxin-antitoxin mixture. Dr. Theobald Smith's suggestion made in 1907 that a mixture of toxin and antitoxin might be useful for human immunization was first employed by Dr. William H. Park in 1913. In the twenty-two years since that time many thousands of children have been immunized with mixtures of this sort. Our experience in Massachusetts has shown that toxin-antitoxin mixture is efficient, safe and in a great many ways entirely satisfactory. It is true that in two communities there were severe reactions following the use of material which had been frozen, but that material was made by a formula now no longer in use and there is no reason to believe mixtures of the present type are subject to increased toxicity when frozen. On the other hand, frozen mixtures may prove to be less satisfactory as immunizing agents and should, therefore, never be used.

Chiefly as a result of some case reports from the middle west, there has been created a fear that toxin-antitoxin mixture will sensitize to horse serum. We have not felt that the sensitization so resulting, if it occurred at all, was of a degree to be practically important. The continued use of toxin-antitoxin mixture during the past two years when diphtheria toxoid has been

*From the Massachusetts Department of Public Health.
Read before the Section of Pediatrics at the One Hundred and Fifty-Fourth Annual Meeting of the Massachusetts Medical Society, Boston, June 5, 1935, as part of a symposium on the control of certain communicable diseases.

†Robinson, Elliott S.—Director, Division of Biologic Laboratories, Massachusetts Department of Public Health. For record and address of author see "This Week's Issue," page 240.

available also shows that this fear is not generally held by the physicians in Massachusetts. For reasons which will be gone into later, our present feeling is that diphtheria toxin-antitoxin mixture remains the most satisfactory immunizing agent for use in adults and in older children, but may well be replaced by diphtheria toxoid in younger children.

About ten years ago Ramon in France introduced the use of diphtheria toxoid, or, as he called it, "anatoxine". This material consists of diphtheria toxin which has been detoxified by the addition of formalin and a period of incubation lasting a month or more. As a result of this treatment, the toxin loses entirely its poisonous properties but retains those properties which give it antigenic value. Extensive experience abroad, particularly in France, but also in Canada and many parts of the United States, has shown that diphtheria toxoid is a satisfactory immunizing agent. The chief objection to its use lies in the fact that older children and adults may have reactions, both local and general, following its use. These reactions are probably due to previous sensitization to the protein of the diphtheria bacillus; and inasmuch as opportunities for sensitization are more numerous the longer one lives, such sensitization is, therefore, more likely to be found in older individuals. When we first distributed this product in Massachusetts, we recommended that it be used only in children under the age of six. Since then we have gradually raised the age limit until now we feel that it may be used with safety in children certainly up to the age of eight or ten and possibly somewhat older. There is some reason to believe that the age limit may be higher in those communities that have had relatively little diphtheria, and that the age limit should be lower in those communities having a good deal of diphtheria. Although we have no figures to support such a statement, it is quite in line with what we know of the disease and of immunization to it. In beginning immunization in a community with diphtheria toxoid, the most logical procedure would be to start with the younger children and as they were done with no reaction gradually increase the ages of the children immunized until reactions became too frequent to be satisfactory. Inasmuch as the reactions are unpleasant or uncomfortable rather than dangerous there would be no harm in such a procedure and it would lead to the immunization of the greatest number of children with diphtheria toxoid rather than with diphtheria toxin-antitoxin mixture. For those children in the higher age groups, toxin-antitoxin mixture should be used.

The number of doses and the intervals between them have been the subject of much discussion. This question is best answered by recalling that the five important factors in producing immunity are the antigen, the size of the dose, the number of doses, the interval between doses, and the basic immunity of the individual; and that we are anxious to produce the highest level of immunity reasonably attainable. We have advocated three doses rather than two because three doses are necessary to attain high levels of antitoxin. The interval of three weeks between doses probably does not give a much larger percentage of Schick negatives than are obtained with intervals of a week; but each individual will have more antitoxin and, therefore, will remain Schick negative longer, because it takes longer for the antitoxin content to fall from the higher value. If only two doses of toxoid are given, the interval should be not less than a month.

Within the past few years there has been a great deal said about alum-precipitated diphtheria toxoid. This product consists of the diphtheria toxoid previously described which has been treated by the addition of alum to give a precipitate. After washing, this precipitate is resuspended in salt solution. The dose is usually 0.5 or 1 cc. The advantage claimed for the use of alum-precipitated toxoid is that only one injection is required and that the level of immunity obtained is at least as high as is ordinarily obtainable with three doses of diphtheria toxoid. The early reports of immunization with alum-precipitated diphtheria toxoid, particularly those from Alabama, indicated that very satisfactory results were secured. Following the experience in Alabama, other manufacturers undertook to produce this product and it has been used rather extensively during the past few years. In many instances the results obtained have been quite in line with the original claims but in other instances results have not been so satisfactory. Unfortunately, certain lots of alum-precipitated diphtheria toxoid have given rise to local reactions which have gone on to the formation of sterile abscesses. In some instances these abscesses have occurred in a large proportion of children inoculated. So far as I know, none of these reactions have left any important sequelae, but such occurrences tend to give a bad reputation to the process of immunization. It is thought that these severe reactions may be traced to one of two causes, both of which lie in the method of manufacture. Some lots which have caused reactions have had a high content of alum. In other instances the fault has been laid to the fact that the toxin was allowed to incubate for a rather long period during the process of toxin formation. This caused the autolysis of an unduly large number of diphtheria bacilli and consequently a high content of diphtheria bacillus protein. One or both of these factors have been involved in those lots which have given unduly numerous reactions, and it is felt that, by avoiding these two faults, reactions may be prevented. A third source of reactions lies in the same mechanism which gives rise to reactions with the ordinary diphtheria toxoid; namely that the in-

dividuals are sensitive to diphtheria bacillus protein and, therefore, react when toxoid is administered in any form. Reactions from this cause can be prevented only by the use of the product in the lower age groups. Such an age limit is perhaps desirable and I understand is in force in New York City, where the use of alum-precipitated toxoid is restricted to children under four years of age. Just so long as diphtheria immunization work is left to school clinics, alum-precipitated toxoid will obviously be of little use. There is every reason, therefore, to continue to urge the immunization of children early in their lives, preferably in the period between six months and one year of age, for at this stage of their development children stand immunization well and respond best with the formation of antibody and, consequently, immunity. Immunization at this age is of course feasible only if it is done by the family physician, for very often he is the only physician who has the opportunity to immunize these children.

The subject of the control of diphtheria and particularly active immunization against diphtheria would not be complete unless mention were made of the use of the Schick test. At the time immunization was first undertaken it was considered desirable to do Schick tests on all individuals and then immunize only those who were positive. Subsequent experience showed that so many of the younger children were positive to the Schick test that it was administratively simpler to omit that operation and immunize all children whose parents gave consent to it. In consequence the use of the Schick test preliminary to immunization has been largely abandoned, except in connection with the study of immunization methods. Its use in such studies is still essential for it is important to know how many children have been immunized by the agent employed and how many were originally Schick negative and therefore unaffected by immunization, at least unaffected so far as can be determined by the Schick test. With the earlier and less perfect immunizing agents, there was also a variable but sometimes large percentage of children who were not immunized in spite of treatment, which necessitated Schick tests following immunization to determine the children needing further injections. Particularly since the introduction of diphtheria toxoid, this retest has been omitted in many instances because the percentage of children immunized is well over 90 and it is felt that the extra effort involved in Schick testing might better be devoted to the immunization of still other children. Such a position is entirely tenable from the standpoint of public health practice and its practical success indicates that there is no particular harm in omitting the subsequent test when all that is desired is to obtain the greatest immunity for the greatest number; in other words, when the problem of diphtheria immunization is looked upon from the public health standpoint, rather than from that of the individual. On the other hand, by means of the retest it is possible to pick out those individuals who need further immunizing injections in order to render them Schick negative and immune to the disease.

One or two words of caution are perhaps desirable in regard to the Schick retest. It has been the practice in some communities to do only the toxin injection, omitting the control. Fortunately, we have always felt in Massachusetts that such tests were not reliable in that they fail to pick out immune individuals with pseudo reactions. The omission of the control injection is particularly bad in doing retests in individuals who have been immunized with diphtheria toxoid, and more particularly with alum-precipitated diphtheria toxoid, since there appears to be considerable sensitization to the constituents of the Schick test material by either of these immunizing agents. Consequently, if only the toxin injection is done, the number of apparent positives will be very much larger than it is in actuality.

A word might be also said about the possibility of an anaphylactic type of reaction following Schick tests done after immunization with toxoid and alum-precipitated toxoid. So far as I know, reactions of this sort did not occur following immunization with diphtheria toxin-antitoxin mixture, but there have been reported now approximately half a dozen cases in which an anaphylactic type of reaction came on immediately after the administration of Schick test material. None of these reported reactions have resulted fatally, but in several of them the reaction was so severe that danger of death appeared imminent. The occurrence of these reactions, infrequent as they have been, strongly suggests that retests should be done only when one is equipped to combat them. The best method of treating such reactions is undoubtedly the use of adrenalin and the application of a tourniquet on the arm above the site of the injection. In at least one instance it apparently was only the toxin injection and not the control injection which caused trouble. There is not sufficient information about the other reported reactions to tell whether one or both injections were at fault.

In closing, it is well to stress again the importance of immunization of children as they appear on the scene. The low incidence of diphtheria during the past few years can only be maintained providing the immunity level of the child population is kept at as high a level as possible. This means that every effort must be continued to secure the immunization of children, and particularly infants, regardless of the fact that there is little diphtheria in many communities. We can hope to keep down diphtheria only by continuing immunization.

CHAIRMAN GARLAND: The papers will now be summarized by Dr. Richard M. Smith.

SUMMARY*

BY RICHARD M. SMITH, M.D.†

AS practitioners of medicine we are called upon to decide a number of practical questions in relation to the prevention of disease. The procedures which have been discussed in the papers this morning and others which have not been considered may be divided into four groups.

1. Those of proved value to be employed for every child.
2. Those of proved value to be used under limited conditions.
3. Those of proved value to be used for individuals exposed to disease.
4. Those of possible efficacy but still in the experimental period.

1. In the first group, procedures of proved value to be employed for every child, we should include smallpox vaccination and immunization against diphtheria. We must decide three matters in connection with these procedures: how can all the children be given protection, when is the best time to do it, and what technique shall be used? The report of the recent White House Conference on Child Health and Protection showed clearly that thus far we have not been successful in giving protection to all children. Among children up to six years of age in the urban communities surveyed, only twenty-one per cent had been vaccinated and immunized and in the rural districts only seven per cent vaccinated and eighteen per cent immunized. It has been assumed that the educational activities of the Health Departments and other agencies would inspire parents to seek protection for their children. Clearly this has not been the case with the majority of parents. A more individual approach is needed. I believe that physicians should take the initiative toward the children under their care. It is felt by some physicians that this is solicitation of practice and thus unethical. I do not agree with this point of view. We take the initiative unhesitatingly in indicating desirable therapeutic measures, indeed we consider ourselves culpably negligent if we fail to do so. I think our responsibility in the field of prevention is equally great.

The time at which immunization and vaccination should be done may vary with individual conditions but, in general, both should have been completed by the time the child is one year old. There is some evidence to indicate that the rare complications following vaccination may be reduced in number or possibly prevented if some other protective inoculation has been given previously. For this reason it is my custom to give the diphtheria immunization at about nine months of age and vaccination when these inoculations have been completed.

This is not the place to discuss the technique of smallpox vaccination. In relation to diphtheria immunization we cannot do better than follow the recommendations of Dr. Robinson, to use three doses of toxoid given at three-week intervals.

2. In the second group, procedures of proved value to be used under limited conditions, we should include for practice in this part of the country scarlet fever and typhoid immunization.

I am fully aware of the fact that some persons would place scarlet fever immunization in group one but there are several reasons why it seems to me we are not yet ready to use this procedure for every child. At the present time, scarlet fever occurs usually in a mild form with only occasional severe complications. Dr. Anderson in his review of the subject has shown clearly the disadvantage of the Dick toxin and the lack of finality in opinion concerning scarlet fever toxoid. I believe it is desirable to await further experimentation, demonstration, and standardization of technique before urging universal applications of this procedure. Under certain conditions there is every reason to expect that satisfactory results from its use may be obtained.

The situation in relation to typhoid fever is somewhat different. The technique is established. The only question is when to employ it. Infants rarely have typhoid fever because their food is usually carefully controlled. Very young children, however, not infrequently acquire the disease. A considerable proportion of cases of typhoid fever seen in this state are infected while they are away from home. It would seem to be a wise practice to give typhoid vaccine to all persons who take food or water under conditions not within their immediate knowledge or control—particularly to those traveling during the summer months.

3. In the third group, procedures of proved value to be used for individuals exposed to disease, the common diseases to be mentioned are measles, chicken pox, rabies, and tetanus. We shall consider only measles.

Dr. Eley has presented the facts concerning the use of various substances to protect from or to modify the severity of measles. I can add

*Presented before the Section of Pediatrics at the One Hundred and Fifty-Fourth Annual Meeting of the Massachusetts Medical Society, Boston, June 5, 1935, as part of a symposium on the control of certain communicable diseases.

†Smith, Richard M.—Assistant Professor of Pediatrics and Child Hygiene, Harvard Medical School and School of Public Health. For record and address of author see "This Week's Issue," page 240.

nothing to what he has said except to call attention to further experimentation which is being carried on to determine the efficacy of placental extract given by routes other than subcutaneous injection. The practical matter which we have to decide is whether to use any substance and if it is to be used, when. Except under unusual individual circumstances, modification of the disease is preferable to protection. Modification should be used for all exposed children who are under three years of age and probably for all under five years of age. If further experience proves that subcutaneous inoculations can be displaced by a method of administration which gives no reaction, it would seem desirable to modify the severity of the disease in all persons exposed to measles.

4. In the fourth group, procedures of possible efficacy but still in the experimental period, we would place immunization against whooping cough. Perhaps one should mention poliomyelitis also, but fortunately it is outside of the field under consideration this morning.

Pertussis is such a dread disease that physicians and parents easily take "the will for the deed" so far as proof in relation to prevention is concerned. Dr. McDonald has been judicial in his conclusions. All of us would do well to follow his lead. Enthusiasm in relation to the value of vaccines has waxed and waned many times. Just now we are in a period of optimism but there have been times in the past when success seemed almost as near at hand as it does to-day and further experience has ushered in a period of depression. One is certainly justified in using vaccine in an attempt to produce immunity against whooping cough but one must be honest with himself and with his patients in acknowledging that proof of its protective efficacy is lacking. One must also guard against drawing conclusions from a few cases, from clinical impressions or from uncontrolled statistical reports. Time will determine whether a vaccine can be prepared of such a quality that, administered according to a given technique, it will afford protection against the disease. Until then let us maintain an open mind.

CHAIRMAN GARLAND: I will now ask for the report of the Nominating Committee.

DR. ALLEN: The Nominating Committee submits the following report:

Chairman for next year, Dr. George P. Hunt, of Pittsfield; and for Secretary, Dr. James M. Baty of Boston.

CHAIRMAN GARLAND: You have heard the report of the Nominating Committee: for Chairman, Dr. George P. Hunt; for Secretary, Dr. James M. Baty. Are there any further nominations? If not, the Chair will entertain a motion for the nominations to close.

[Voted, that nominations be closed.]

CHAIRMAN GARLAND: The Chair will entertain a motion for the Secretary to cast one ballot for these officers.

[VOTED, that the Secretary cast one ballot for the officers as nominated.]

The Secretary cast one ballot for the officers nominated, and they were declared duly elected.

CHAIRMAN GARLAND: The papers are open for discussion. I will ask the discussers to give their names and addresses plainly, and remember that discussion is limited to two minutes.

DR. CONRAD WESSELHOEFT, Boston: In regard to the use of convalescent serum in measles, I would like to say a word from the standpoint of one who holds the pursestrings of one supply of this convalescent measles serum. It is not a pleasant position to hold because some people have the idea that measles is such a dread disease that it is very dangerous, even for an adult, and some family physicians explain to their adult patients that in the event of exposure and no knowledge of having had measles, if they could get some of this convalescent measles serum they would not come down with measles. Of course, the bread-winner of the family does not want to contract the disease, and pressure is brought to bear on some of us who hold the pursestrings of this convalescent measles serum. If he happens to be a hospital trustee then I am up against it. I would like to say that it takes at least four times as much of this very valuable convalescent measles serum to protect a full-grown trustee as it does to protect a small child. The difficulty in obtaining the serum lies in the fact that it is obtained only from adults, and few adults have measles.

I would like to emphasize the ruling that has been made in Providence, where they do not release the convalescent measles serum for any case that is not under four years old, except under those conditions which Dr. Eley brought out; for instance, if a child has whooping cough or is convalescent from scarlet fever. Then, of course, it is very desirable that the measles serum should be used.

I think the general tone to-day has been to stress the severity of these diseases. I think it is overstressed. The high mortality in measles comes before the age of four. After that it is not, according to the statistics, a dread disease. So I welcome, and so does everybody welcome, a placental extract which is so effective, and which may do away with this demand for convalescent serum which is far greater than the supply. At contagious hospitals a supply of this convalescent measles serum has to be kept on hand because measles can very well break out in a scarlet fever ward.

I would like to say in connection with this that I am always very much surprised when a doctor sends to the Haynes Memorial a case of scarlet fever knowing perfectly well that that child has also been exposed to measles within a week, and yet says nothing whatever about it at the time he calls up to admit the case. It is astonishing how often that happens.

As to scarlet fever, we must all welcome this toxoid because we know that we do get unpleasant results from the use of the toxin in the usual doses, or even though we split them up, as I do, into eight doses over eight weeks, and even then we sometimes get reactions. The toxoid would seem to do away with that.

Just one more point here, and that is that those of us who are interested in scarlet fever are very much concerned about this Dick patent. Of course, the discovery of the toxin was an important step, as has been brought out by Dr. Anderson. The active immunity obtained from this toxin not only protects against the disease, but it protects against the complications as well, because pupil nurses so protected by inoculations when intimately exposed on scarlet fever wards not only fail to come down

with scarlet fever but they also do not come down with tonsillitis, otitis media, and all the other complications arising from the pyogenic property of this hemolytic streptococcus. On the other hand, the patient in the early stage of scarlet fever who is given the antitoxin may be immediately and dramatically benefited for the time being but is not thereby protected from the subsequent complications. Our present conception of the origin and nature of all these complications is very unsatisfactory.

The trouble with the Dick patent is that it not only covers the discovery of the toxin, but all the discoveries which lead up to it; and, furthermore, the patent is so all-embracing as to prohibit anyone from inoculating, with any scarlet fever toxin, any human being or animal who is not licensed by the Scarlet Fever Commission in Chicago. The patent was sanctioned by a committee of the A.M.A. with the idea that it would protect the profession from bad products during the period of its introduction. That was ten years ago. Such protection is no longer needed, if, indeed, it ever was needed. The Massachusetts Medical Society in Council yesterday passed a resolution that this Society was opposed to the continuation of that patent, and the delegates are to bring that resolution before the American Medical Association.

CHAIRMAN GARLAND: Is there any other discussion?

DR. H. B. MARBLE, Shelburne Falls: I have just three questions I would like to ask. The first one is: What is the present status of the value of ether injection in the treatment of whooping cough? The second, What would be a suggested dose or an average dose of toxoid in pupils of high school age? The third one is, we all know there are many children in the high schools who have been vaccinated, but not successfully. What can be done about getting these children vaccinated so that they will be immune to smallpox?

CHAIRMAN GARLAND: Any further questions?

DR. EDWARD M. SMITH, Salem, New Hampshire: I wish to call attention to the importance of the mammalian transition period after birth and at the commencement of nursing. I believe that some of our pediatric problems along the line of discussion begin here. They can be solved or dismissed without understanding by the use of the neglected substance, the placenta.

The physiological effects and biological substances in the after-birth are too challenging for further neglect. (1) The fascinating McKhann immune bodies have been discussed and they or their precursors may be transmitted from the milk of the placenta-fed, or placenta-extract-enriched postpartum woman. (2) Not common knowledge is the fact that placenta has been shown to be one half more valuable per weight than the best liver in blood remitting substance, or what I shall call Carnivorous Substance X (verbal communication, Dr. William Castle, 1935). (3) Estrogenic substance is present in highest titre in this embryonal material. It has definite lactogenic effects even on the male, and one of the best tests for its presence is the growth of nipple and areolar skin. It may cornify and change the whole mucosa of the genital tract within forty-eight hours. It has been used to secure resistance to juvenile gonorrhea. It should be carefully tried in attempts to render the postpartum state more promptly non-invadable. (4) The hemorrhagic diseases of the newborn with an incidence of 1.6 per thousand may possibly be dismissed by placenta extract directly or through the mother. This erythroblastic crisis responds to liver extract and the naturally available substance should be preferred. Its explosive potentiality in stimulating and homeostatic balancing of mitosis is well known by the typical Minot-Murphy reticulocyte curves. (5) The presence in high titre of progestin requires observation of the control over involution and subinvolution of the uterus. (6) The nonspecific substances present in both liver extract, placenta, and placental extract which extends to cord and brain changes in primary anemia may reasonably be expected to disturb postnatal nutrition and render breast feeding more vitally valuable. The effect needs study in the jaundice of the newborn, the immunity of the newborn, the early and later mortality of the newborn. It certainly may be used in the primary anemia of pregnancy.

Lying-in hospitals, as my own, the Boston Lying-In, are to date disposing of as garbage, this most valuable physiological material. It should be saved, extracted, and rendered generally available for the studies indicated. About 3,000 lbs. is lost in this hospital alone.

Mammalian survival has been largely conditioned by "the conservation of all already elaborated material." Placental eating and the resorption of the young are common indicators of nature's extreme conservatism in a mammalian economy. The discovery of carnivorous substance X (blood important) is partly based on the observation of carnivorous conservation of most valuable elaborated substance.

Important questions are suggested by my remarks with respect to correction of past errors in the treatment of the newborn child and the mother.

CHAIRMAN GARLAND: Any further discussion? Dr. Eley, have you anything to add?

DR. ELEY: No.

CHAIRMAN GARLAND: Dr. McDonald? Perhaps you can answer this question as to the use of ether and oil.

DR. MCDONALD: Dr. Smith perhaps could give a more complete answer to the question concerning the use of ether injection in the treatment of whooping cough. I have had no experience with its use. Local practitioners seem to agree that the average case of whooping cough will respond best to rest and that it is only the rare case that requires the use of ether. An appraisal of the literature gave me an unfavorable impression for its continued use in the average case.

CHAIRMAN GARLAND: Dr. Anderson?

DR. ANDERSON: I haven't anything further to add in regard to scarlet fever. I would take the liberty of answering the Doctor's question about the vaccination certificates to high school pupils. As I understand your situation, the child is in the high school, having not been successfully vaccinated. The Supreme Court of Massachusetts has ruled that the school committee is within its rights in requiring a parent to renew a vaccination examination certificate as often as once every two months. There has never been any ruling, however, on how often an attempt at vaccination shall be made, but many school committees have interpreted that to the point of requiring that the vaccination attempt be made each year until such time as a successful take shall have been obtained. That has not, however, been ruled upon by the Court; it is merely precedent, and, as far as I know, has never been challenged.

CHAIRMAN GARLAND: Dr. Robinson?

DR. ROBINSON: Only to answer the question about giving the toxoid to high school children, and the answer to that is that the only way you can tell is by trying it. I would suggest a dose not over .16 cc. and see what happens, then perhaps doubling it each time.

CHAIRMAN GARLAND: Dr. Smith, have you anything to add?

DR. RICHARD M. SMITH: Nothing.

CHAIRMAN GARLAND: Is there any further discussion? If not, I declare the meeting adjourned.

PROCEEDINGS OF THE SOCIETY

First Day, June 3, 1935

THE one hundred and fifty-fourth anniversary was observed in Boston, Monday, Tuesday and Wednesday, June 3, 4 and 5, 1935. The headquarters were located at the Hotel Statler and, with the exception of the clinics, all the meetings were held in this Hotel.

On Monday morning an interesting series of motion pictures was shown in the Georgian Room. At 9:15 the new Section of Dermatology and Syphilology held its first meeting in the Ball Room Assembly with an attendance of one hundred.

Monday afternoon the Section of Obstetrics and Gynecology met in the Georgian Room with an attendance of 450 and the Section of Radiology and Physiotherapy met in the Ball Room Assembly with an attendance of fifty.

Monday evening the Shattuck Lecture was delivered by Dr. William E. Gallie of Toronto on the subject of "Sprains and Dislocations." This paper appeared in the *New England Journal of Medicine*, issue of July 18. Following the Shattuck Lecture Dr. Charles H. Tozier showed some interesting colored pictures and motion picture films as a part of his talk on "The Land of the Aztecs." Light refreshments were served at the close of the meeting.

Second Day, June 4, 1935

Tuesday morning the Section of Surgery met in the Ball Room Assembly. There were 300 in attendance. Medical Clinics were offered at the Massachusetts General Hospital. The Supervising Censors met at 11:30 and the Council assembled for its annual meeting in the Georgian Room at 12:00 o'clock. There was a recess for the Cotting Luncheon following which the business meeting was resumed.

In the afternoon the Section of Medicine met in the Ball Room Assembly with an attendance of 450.

Tuesday evening at 7:00 o'clock the Annual Dinner was served in the Georgian Room. There were approximately 304 people in attendance. President Robey acted as toastmaster and introduced the speakers with appropriate remarks. The speakers were: The Honorable Frederick W. Mansfield, Mayor of Boston; Dean Roscoe Pound of the Harvard Law School; the Honorable Thomas H. Buckley, representing His Excellency, the Governor; Dr. Douglass V. Brown, Assistant Professor of Medical Economics, Harvard Medical School and the Reverend Phillips E. Osgood of Emmanuel Church. The President then presented President-Elect Charles E. Mongan of Somerville who greeted the Fellows and guests and spoke briefly on some of the economic problems which confront the Society. The meeting adjourned at 10:40 P.M.

Third Day, June 5, 1935

The Section of Pediatrics held its meeting in the Georgian Room with 325 in attendance. The Section of Tuberculosis met in the Ball Room Assembly with an attendance of 150. At the Massachusetts General Hospital the Staff presented a Surgical Dry Clinic.

The Annual Meeting of the Massachusetts Medical Society was held in the Georgian Room at 12:00 o'clock noon. The President of the Society, Dr. William H. Robey, presided and Dr. A. S. Begg was present as Acting Secretary.

After calling the meeting to order the President asked for the reading of the records of the last meeting which was held in Worcester on June 6, 1934. There being no changes, he declared the record approved.

The Acting Secretary then presented figures showing the changes in membership:

Membership Reported June 6, 1934			4951
Deduction because of late compliance with rules			2
			4949
Losses			
Deaths	103		
Resignations	19		
Deprivations	62		
		184	
Gains			
New Fellows	243		
Readmitted by Censors	1		
Restored by Council	7		
		251	
Net Gain		67	67
Membership Reported June 5, 1935			5016

The President then inquired for any resolutions which were to be offered at this Annual Meeting. There being none he proceeded to

present the delegates from other State Societies in New England.

Dr. Forrest Bertram Ames of Bangor, Maine, complimented the Society upon the excellence of the papers presented and stated that his message from the Maine Medical Association consisted of a cordial invitation to the Fellows to attend the Annual Meeting of the Maine Medical Association at York during the last week in June.

Dr. M. E. Rindge of Madison, Connecticut, paid his compliments to the Committee of Arrangements and extended the greetings of the Connecticut State Medical Society to the Massachusetts Medical Society.

Dr. Robert B. Greenough of Suffolk then moved that the Society go into executive session and that all those persons who were not Fellows of the Society, with the exception of the stenographer, be excluded from the hall. The motion was seconded and carried. Dr. Robey then declared the Society to be in executive session and proceeded to call for the report of the Board of Trial. After an extended discussion it was voted to accept the report of the Board of Trial and to refer it to a special committee consisting of the Presidents of the District Societies and of the Massachusetts Medical Society of which the President of the Society shall be chairman. This committee shall consider the findings of the Board of Trial and shall recommend to the Society at its next Annual Meeting such action as seems to it just and necessary.

There being no further business to come before the meeting, President Robey proceeded to read his address, which was published in the *New England Journal of Medicine,* June 27, 1935. The meeting then adjourned for luncheon.

The meeting was again called to order at 2:00 P.M. Dr. Robey stated that Dr. Brace Whitman Paddock of Pittsfield, Mass., was elected Orator at the Worcester meeting last June and that he had completed the preparation of his oration just prior to his death on May 22 of the present year. Inasmuch as Dr. Paddock's son, Mr. Frank K. Paddock, is engaged in the study of medicine, it seemed fitting to ask this gentleman to read the address written by his father.*

The meeting adjourned at 3:15 P.M.

A golf tournament was held at the Belmont Springs Country Club in the afternoon.

ALEXANDER S. BEGG, M.D.,
Acting Secretary.

It was a matter of frequent comment by the Fellows in attendance that the work of the Committee of Arrangements had been of an exceptionally high order. The topics on the section programs were well chosen and the arrangements to handle the large number of visitors worked smoothly and without confusion. The assignment of booths for the Commercial Exhibits was highly satisfactory and the Scientific Exhibits were so placed as to give an opportunity for the Fellows to observe the recent advances in Medicine.

A unique feature of this meeting was the special program for ladies which was arranged by the Committee consisting of Mrs. William H. Robey, Chairman; Mrs. Roger I. Lee, Vice-Chairman, and Mrs. Herrman L. Blumgart, Mrs. Frederick L. Good, Mrs. Elliott P. Joslin, Mrs. George R. Minot, Mrs. George P. Reynolds, and Mrs. William M. Shedden. The attendance was unusually large but was capably handled by the ladies who were asked to assist the committee. The calendar was as follows:

MONDAY, JUNE 3.

Registration, Hotel Statler Mezzanine.

1. Tea—from 4-6, at the home of Mrs. Randall Clifford, "Green Hill", 215 Warren Street, Brookline.

TUESDAY, JUNE 4.

Registration.

10-12—Visit to Social Service, Occupational Therapy and other departments of Massachusetts General Hospital.

2. Tea—The Isabella Stewart Gardner Museum, Fenway Court. Mr. Morris Carter, Director, graciously arranged for a personally conducted tour from 3-4 P.M. Reception at 4 o'clock and tea at 4:30.

3. Buffet Supper with entertainment—6:30 P.M. Junior League, Zero Marlborough Street.

4. Trip to Concord.
   Lunch for the ladies at Concord Country Club, and the Old Mill Dam, as guests of the Massachusetts Medical Society, in honor of the wives of the Presidents of the District Medical Societies.
   Visit to Mrs. Russell Robb's garden.

The total registered attendance at the three-day meeting was 1342. The attendance at each of the Section meetings and the officers elected for 1936 follow:

SECTION OF MEDICINE. Attendance, 450.
Chairman, William D. Smith, Brookline and Boston.
Secretary, Laurence B. Ellis, Belmont and Boston.

SECTION OF SURGERY. Attendance, 300.
Chairman, E. Parker Hayden, Boston.
Secretary, Frederick S. Hopkins, Springfield.

SECTION OF TUBERCULOSIS. Attendance, 150.
Chairman, Donald S. King, Boston.
Secretary, Olin S. Pettingill, Middleton.

*Published in the issue of June 6, page 1069.

SECTION OF PEDIATRICS. Attendance, 325.

Chairman, George P. Hunt, Pittsfield.
Secretary, James M. Baty, Belmont and Boston.

SECTION OF OBSTETRICS AND GYNECOLOGY. Attendance, 450.

Chairman, Charles J. Kickham, Brookline.
Secretary, Raymond S. Titus, Boston.

SECTION OF RADIOLOGY AND PHYSIOTHERAPY. Attendance, 50.

Chairman, Philip H. Cook, Worcester.
Secretary, William G. Curtis, Wollaston.

SECTION OF DERMATOLOGY AND SYPHILOLOGY. Attendance, 100.

Chairman, Harvey P. Towle, Boston.
Secretary, Rudolph Jacoby, Boston.

ADMISSIONS RECORDED FROM JUNE 6, 1934 TO JUNE 5, 1935

Year of Admission	Residence	Medical College
1934	Abrams, Jacob Irving, Boston	10
1935	Adams, Herbert Dan, Newton Center	11
1935	Alemian, Arsham, Weymouth	12
1935	Alexander, Kirke L., Orange	10
1934	Amaral, Francis Medeiros, Worcester	10
1935	*Amaral, Manuel Francis, Cambridge	6
1935	Amesbury, Ivon Cuthbert Raleigh, Dorchester	10
1935	Arrowood, Julia Gordon, Worcester	10
1934	Bain, William Joseph, Jr., Lawrence	12
1934	Baker, Harry Abraham, Holyoke	12
1935	Baker, Robert Henry, Worcester	32
1934	Barker, John Russell, Newtonville	12
1935	Barnes, Wayne Clifton, Springfield	12
1934	Barrett, Thomas Gerard, Marblehead	12
1934	Bassow, Carlton Francis, Athol	7
1934	*Bavuso, Anthony Christiana, Framingham	6
1935	Beers, Sherman Johnson, Plymouth	1
1934	Benjamin, Louis Burton, Weymouth	11
1934	Bennett, Darwin Elbridge, Fitchburg	11
1934	Berg, Carl, Millis	12
1935	Bergin, Paul Francis, Worcester	10
1934	Bianco, Harvey Henry, North Adams	19
1935	Black, Muriel Gene, Dedham	10
1934	Blank, Edward Arthur, Brookline	10
1934	Bombaci, Augustine, Medford	12
1935	Borkovic, Embrie John, Worcester	34
1934	Bourke, Robert William, East Walpole	12
1935	Bray, Thomas Ambrose, Worcester	19
1934	Bray, Walter Abner, Essex	12
1934	*Brenner, Nathan, Roslindale	6
1934	Brewer, James Franklin, Jr., New Bedford	3
1934	Brody, Myer, Braintree	10
1935	Budnitz, Edward, Worcester	11
1934	Burckel, Arthur Walter, Worcester	10
1934	Butler, Glenn Vaughn, Weymouth	10
1934	Callanan, Eugene Francis, Boston	12
1934	Campbell, Frederick Leon, Brighton	12
1934	Cantor, Harold, New Bedford	12
1935	Carrano, Armand T., Fitchburg	29
1935	Cauley, John Henry, Dorchester	12
1935	Chadwell, Kenneth James, Lynn	10
1935	Chapman, Earle MacArthur, Brookline	18
1935	Chernoff, Arthur Hylan, Middleton	12
1935	Chittick, Rupert Addison, Foxborough	11
1935	Claiborne, Thomas Sterling, Boston	35
1935	Clark, Theodore Dodge, Newtonville	11
1934	Clarke, Henry Gisler, Northampton	11
1934	Cohen, Lionel, Holyoke	9
1935	Cohen, Sidney Slater, Brookline	11
1934	Cole, Edwin Muncks, Cambridge	11
1934	Collins, John Francis, Revere	19
1935	Conlogue, Everett Freeman, Wakefield	10
1935	Coughlin, Edward Joseph, Williamstown	29
1935	Craig, Henry Roosevelt, Worcester	36
1935	Crawford, George Marshall, Boston	25
1934	Crumb, Harold James, Lexington	12
1935	Cummings, Charles Stearns, Onset	10
1934	Davenport, Lowrey Frederick, Waltham	5
1934	Davis, Ada Frances, Merrimac	10
1935	Denovian, John, Belmont	29
1934	Di Bona, Astro Attila, Cambridge	12
1935	Di Clerico, Joseph Anthony, Nahant	19
1934	Donovan, Robert Joseph, Brookline	10
1935	Doyle, George Melvin, Gloucester	18
1934	D'Urso, John Joseph, Lawrence	11
1935	Egan, Christopher Edward, Gloucester	19
1934	Ellis, Sydney, Boston	12
1934	Ellms, Evelyn Blanche, Tewksbury	12
1934	Erickson, George Carl, Worcester	12
1935	*Ettinger, Alice, Boston	37
1934	Faraci, Philip, Haverhill	12
1934	Feigenbaum, Jacob, Allston	9
1934	Ferrant, Michael Edward, Boston	12
1934	Finn, Herbert George, Charlestown	11
1935	Flanders, Walter Herbert, Melrose	10
1934	Flynn, Joseph Edward, Boston	1
1935	Ford, Carlton S., Attleboro	12
1935	Frazee, John Richard, Brighton	11
1934	Frumson, Soloman Louis, Boston	12
1935	Gagliardi, George Richard, Milford	10
1934	Gallagher, James, Roswell, Andover	1
1935	Gavolio, Frank Joseph, Waltham	12
1934	Gazzaniga, Dante Achilles, North Adams	42
1934	Goldenberg, Harry, Boston	12
1935	Goldstein, Henry, Medford	12
1935	Goodman, Melvin, Hathorne	12
1935	Goodwin, Paul, Clinton	19
1935	Goodwin, Robert Hancock, Assonet	11
1935	Gorman, Arthur Joseph, Boston	12
1935	Gugenberger, Joseph, Worcester	39
1935	Hahn, Joseph, Boston	12
1935	Hall, Marshall Goldthwait, Jamaica Plain	29
1935	Hamilton, Harold Henry, Plymouth	11
1934	Hanley, Francis Joseph, Whitman	11
1935	Hanrahan, Arthur Lawrence, Quincy	10
1935	Hardy, Harriet Louise, East Northfield	33
1935	Harpin, Raymond A., Lynn	10
1934	Hart, Clarence Dunbar, West Somerville	13
1935	Helgesson, Uno Helmer, Northampton	1
1935	Hewitt, Wright Platt, Quincy	11
1934	Hilton, Philip Frederick, Waltham	12
1935	Hirst, Elizabeth, Melrose Highlands	10
1935	Hopkins, Henry Perkins, Chatham	9
1934	Hopkins, John Renton, Tewksbury	11
1935	Hyatt, Gilbert Thomas, Fall River	11
1935	Jacobs, Herbert, Springfield	40
1934	Jacobs, Salvador, Hathorne	12
1934	Jaffee, Isadore Harry, Brighton	10
1934	Jaworski, Charles John, Worcester	8
1935	Johnson, Milton Evald, Attleboro	12
1935	Jones, Theodore Winslow, Pittsfield	3
1935	Joslin, Allen Proctor, Boston	12
1934	Joyce, Charles Coniff, Hathorne	7
1934	*Katz, Albert, Chelsea	14
1935	Keith, Theodore Kent, Newton Center	10
1935	†Kelley, Julius Goddard, Pocasset	12
1934	Kelly, Daniel James, Adams	12
1934	Kelly, Joseph Nicholson, Orleans	10
1935	Kendall, Lee Gordon, Framingham Center	11
1935	Kennedy, Joseph Louis, South Boston	12
1934	Kimberly, Charles Hazard, Stockbridge	11
1934	Klapper, Claude, Haverhill	11
1934	*Klickstein, Maurice, Medford	6

1935 Knight, John Ellis, West Roxbury 11
1934 Kobes, Herbert Richard, New Bedford 11
1934 Kosciolek, Stephen Frank, Lowell 10
1934 *Kraft, Meyer Louis, Chelsea 6
1935 Krakower, Abram, Buzzards Bay 9
1934 Krausz, Marguerite Fox, Brighton 12
1934 Kudish, Benedict, Somerville 12
1934 Latham, David Ainsworth, Lowell 9
1934 Latham, Ernest Hargreaves, Lowell 9
1934 Lavoie, Aurel Gilbert 12
1934 *Lawlor, James Joseph, Cambridge 6
1934 *Leavitt, Thomas William, Malden 20
1935 *Lenares, Joseph Edward, Milford 6
1935 Liberman, Charles, Winthrop 12
1935 Lingley, James Reginald, Belmont 11
1935 Lough, Norbert Francis, Rockland 10
1934 Lundy, Joseph Augustine, Worcester 12
1935 Lyons, Timothy Francis Patrick, Dorchester 12
1934 MacCready, Robert Alvan, Williamstown 11
1935 Mackie, Thomas Turlay, New York City 29
1935 MacNeil, Melvin Joachim, Waltham 41
1935 Mahlowitz, Alexander, Boston 11
1934 Maletz, Leo, Hathorne 12
1935 Margulois, Benjamin, Dorchester 43
1934 McClelland, Willis Barr, Needham 10
1935 McClintock, Walter Lowrie, Brookline 21
1935 McDonald, Francis C., Boston 11
1934 McGinnis, George Hugh, Allston 12
1934 Militzer, Raymond Edgar, Wrentham 11
1935 Milne, Gordon Charles, Auburn 12
1934 *Mintz, Emanuel Ross, Brookline 16
1935 Mitiguy, Edmund Reeves, Forest Hills 7
1934 Moloney, William Curry, Jamaica Plain 12
1935 Moore, Dorothea May, Cambridge 18
1934 Muellner, Salomo Richard, Brookline 12
1934 Murray, Arthur Carpenter, Winthrop 11
1934 Nakashian, Meran, Ludlow 12
1935 Nasse, John Thomas, Millis 10
1935 Neff, Walter Scott, New Bedford 21
1934 Nelson, Carl Olaf, West Medway 10
1934 Nersessian, Aznive, Foxborough 46
1934 Newell, John Louis, Boston 11
1934 Newmark, Nelson, Springfield 1
1935 *O'Brien, Daniel Jalm, Winthrop 20
1935 O'Connell, John Joseph Jr., Dorchester 12
1935 O'Leary, Cornelius Joseph, Brighton 12
1934 O'Leary, Joseph James, Haverhill 11
1934 Paine, David, Worcester 2
1935 *Persing, William Emanuel, West Stockbridge 6
1935 Petrone, Othilia Vieira, Fall River 12
1934 *Phillips, Richard Betts, Jamaica Plain 17
1934 Pilcher, Lewis Stephen 2nd, Newton Center 11
1935 Polisner, Saul Richard, Brookline 10
1934 Pomerleau, Rodolphe Joseph Firmin, Lowell 10
1935 *Poplack, Samuel Lewis, Taunton 6
1934 Poppen, James Leonard, Boston 15
1935 Potito, Dominic, East Boston 12
1935 Proger, Samuel Herschel, Boston 24
1935 *Quinlan, Joseph David, Fitchburg 6
1935 Quinn, William Henry, Jr., Westborough 8
1934 Rafferty, George William, Boston 11
1934 Ravit, George Jonathan, Brighton 21
1934 Richardson, John Robert, Brookline 11
1934 Rogers, Edward Saunders, Newton Center 11
1934 Rogers, Howard Wooding, Newburyport 29
1934 Rothman, Maurice Samuel, Boston 12
1934 Rouillard, Francis, Brookline 11
1934 Russell, Horace Pond, Rockland 10
1935 Sabia, Marion Corleto, East Boston 12
1935 Sanderson, Robert, Dedham 11
1935 Sartwell, Philip Earl, Middleton 10
1935 *Schatzki, Richard, Belmont 37
1935 Schiller, Irving William, Dorchester 12
1935 Schube, Purcell George, Mattapan 44
1935 *Schwartz, Theodore, Springfield 6
1935 Segal, Maurice Sidney, Boston 12
1934 *Sena, Vincent William, West Somerville 6
1935 Shain, Joseph Herman, Boston 12
1934 Shedd, Charles Gale, Boston 11
1934 Shepardson, Robert Batchelder, Reading 10
1934 Sher, David Daniel, Boston 10
1934 Sherman, Allen Irving, West Newton 12
1934 Shpiner, Leonard Benjamin, Cambridge 15
1935 Sieracki, Louis Anthony, Norfolk 23
1935 Sisson, Milton Melvin, Greenfield 12
1934 Slattery, John Joseph, Medfield 10
1935 Smith, Lawrence Jackson, Springfield 21
1935 Spangler, Huston Kiefer, Worcester 4
1935 Spencer, Jack, Boston 35
1934 Stagg, Arthur Ritchey, Medfield 28
1934 Starbuck, Amber Angelia, Springfield 10
1934 Steinecke, Olga Emma, Taunton 22
1934 Sturgis, George Putnam, Brookline 11
1935 Sullivan, Margaret Mary, Fall River 45
1935 Sullivan, Raymond Francis, Worcester 9
1935 Swinton, Neil Williams, Newton Center 38
1935 Tanner, Walter Lewis, Springfield 10
1935 Tartakoff, Joseph, Jamaica Plain 12
1935 *Taylor, Lewis, Greenfield 6
1934 Teel, Harold Morrison, Roxbury 11
1934 Thomas, Jackson Mash, Waverley 24
1934 Tidmarsh, Clarence Johnson, Waban 9
1935 Townsend, Willis Merrick, Melrose Highlands 10
1934 Travers, Frank, Barnstable 10
1934 Tully, George Chandler, Worcester 7
1934 Udelson, Barnet Arthur, Dorchester 12
1935 Varden, Leo, Attleboro 47
1934 Violette, Roger Norman, Fall River 9
1934 Wails, James Otto, Ayer 27
1934 Walcott, Charles Folsom, Chestnut Hill 11
1935 Walker, Burnham Sarle, Cambridge 10
1935 Wegner, Walter Robert, Boston 26
1934 Whalen, Kearons James, Jr., Pittsfield 30
1934 White, J. Louis, Barnstable 31
1935 Wilkins, George Franklin, Boston 11
1935 *Wilkinson, William D., Boston 6
1934 Wilson, James Leroy, Roxbury 11
1934 Wisner, Frank Bradley, Ludlow 1
1935 Wondolowski, Joseph Peter, Worcester 19
1934 Woodall, James Martin, Jamaica Plain 11
1934 Zanfagna, Philip Edward 10

*Indicates that the candidate's diploma was approved by the standing Committee on Medical Education and Medical Diplomas, he was given a personal interview by this committee and permitted to take an examination before a Board of Censors.

†Readmitted by Censors.

Total = 244

KEY TO MEDICAL COLLEGES

1. Yale University School of Medicine
2. Syracuse University College of Medicine
3. Vanderbilt University School of Medicine
4. University of Pennsylvania School of Medicine
5. University of Rochester School of Medicine
6. Middlesex College of Medicine and Surgery
7. University of Vermont College of Medicine
8. Hahnemann Medical College and Hospital of Philadelphia
9. McGill University Faculty of Medicine
10. Boston University School of Medicine
11. Harvard University Medical School
12. Tufts College Medical School
13. Albany Medical College
14. Chicago Hospital College of Medicine
15. Rush Medical College of the University of Chicago
16. Université de Paris Faculté de Médecine
17. University of Edinburgh Faculty of Medicine
18. Johns Hopkins University School of Medicine
19. Georgetown University School of Medicine

20. College of Physicians and Surgeons, Boston
21. Jefferson Medical College of Philadelphia
22. Medical College of Virginia
23. Indiana University School of Medicine
24. Emory University School of Medicine
25. Northwestern University Medical School
26. University of Nebraska College of Medicine
27. Tulane University of Louisiana School of Medicine
28. College of Medical Evangelists
29. Columbia University, College of Physicians and Surgeons
30. University of Buffalo School of Medicine
31. New York Homeopathic Medical College and Flower Hospital
32. University of Tennessee College of Medicine
33. Cornell University Medical College
34. University of Pittsburgh School of Medicine
35. University of Virginia Department of Medicine
36. University of Georgia Medical Department
37. University of Berlin
38. University of Michigan Medical School
39. Long Island College Hospital
40. University of Chicago School of Medicine
41. Dalhousie University Faculty of Medicine
42. Loyola University School of Medicine
43. St. Louis University School of Medicine
44. University of Cincinnati College of Medicine
45. University and Bellevue Hospital Medical College
46. Temple University School of Medicine
47. George Washington University Medical School

DEATHS REPORTED FROM JUNE 6, 1934, TO JUNE 5, 1935

Admitted	Name	Place of Death	Date of Death	Age
1925	Adam, John Geikie	Great Barrington	December 1, 1934	56
1891	Arnold, Horace David	Waltham	March 11, 1935	72
1895	Baldwin, Herman Trost	Orlando, Florida	November 25, 1934	66
1934	Bavuso, Anthony Christiana	Framingham	December 30, 1934	32
1904	Bellamy, William Woolsey	Watertown	January 17, 1935	57
1924	Bigelow, George Hoyt	Framingham	December 3, 1934	44
1895	†Bradford, Cary Carpenter	Southbridge	October 20, 1934	79
1890	Bragdon, Horace Elwood	Winthrop	February 17, 1935	67
1886	Burrage, Walter Lincoln	Brookline	January 26, 1935	74
1928	Caldwell, John O.	Sharon	January 19, 1935	49
1908	†Chisman, Eudora Pierce (Higgins)	Boston	August 22, 1934	70
1907	Coffin, Frank Herbert	Haverhill	April 7, 1935	58
1922	Conway, Francis Bernard	Boston	September 17, 1934	64
1893	†Coolidge, John Nelson	Ottawa, Canada	January 9, 1935	68
1911	Coupal, James Francis	Washington, D. C.	January 3, 1935	50
1891	Cram, John Wesley	Colrain	April 5, 1935	76
1897	Crowley, Jeremiah Francis	Adams	July 4, 1934	62
1924	Curley, George Frederick	Milford	April 15, 1935	62
1902	Dame, Fred Russell	Athol	December 21, 1934	62
1916	Day, Edward Philip	Dorchester	October 31, 1934	66
1921	Dobson, Clarence Henry	Brookline	February 1, 1935	65
1897	Dow, George Farwell	Reading	December 9, 1934	65
1885	Durant, Charles Edwin	Haverhill	January 19, 1935	72
1906	Ellam, Herbert William	Melrose	June 4, 1935	55
1932	Fennessy, William Casey	Buzzards Bay	June 22, 1934	57
1902	Fisher, Irving Jewell	Liverpool, England	August 30, 1934	57
1894	Freeman, Franklin Willard	Lynnfield Centre	December 5, 1934	74
1900	Gardner, Harrie	Milton	May 11, 1934	64
1887	†Gilbert, John	Fall River	November 3, 1934	87
1928	Girardi, Victor Joseph	Plainfield	August 2, 1934	35
1903	Goodall, Harry Winfred	Boston	April 17, 1935	58
1911	Hall, Gardner Wells	Boston	April 21, 1935	59
1925	Hall, George Morris	Jamaica Plain	March 5, 1935	43
1899	†Hamilton, Annie Lee	Jamaica Plain	September 23, 1934	70
1900	Healy, Daniel Laurence	Framingham	June 21, 1934	62
1912	Hemeon, Frederick Chipman	Dorchester	May 26, 1935	65
1929	Herman, William J.	Boston	January 25, 1935	43
1923	Herrin, Herbert Eliot	At Sea	July 16, 1934	51
1894	†Hodges, Almon Danforth	Roxbury	October 17, 1934	69
1920	Hoey, Warren Henry	Newton Upper Falls	December 31, 1934	53
1916	Holzman, Joseph	Roxbury	June 3, 1935	64
1919	Horr, Albert Winslow	Watertown, New York	August 3, 1934	69
1904	Hussey, Edward John	Holyoke	December 4, 1934	61
1919	Hutchinson, Charles William	Boston	June 26, 1934	49
1905	Jennings, Curtis Herman	Fitchburg	December 31, 1934	58
1884	†Johnson, Francis Emerson	Erving	April 30, 1935	77
1905	Jones, Frederick Elmer	Weston	December 21, 1934	64
1921	Karcher, Edward Winslow	Lynn	February 18, 1935	52
1900	Kepler, Charles Ober	Brighton	November 1, 1934	66
1902	Knight, Charles Lewis	Boston	April 18, 1935	60
1930	Knowles, Charles Augustus	Dorchester	January 31, 1935	30
1916	Lane, Elwin Dexter	Andover	March 25, 1935	58
1897	Larrabee, Ralph Clinton	Boston	March 9, 1935	64
1923	Loughlin, John Joseph	Wakefield	September 6, 1934	54
1914	Lyman, Henry	Ponkapoag	June 15, 1934	55
1889	MacArthur, George Elden	Ipswich	April 10, 1935	77
1893	†Mangan, John Joseph	Lynn	March 29, 1935	77
1917	†Martin, George Forrest	Lowell	November 4, 1934	71

1913	Martin, Harold Winthrop	Milton	April	15,	1935	47
1897	McCabe, John Joseph	Holyoke	November	13,	1934	61
1904	McGillicuddy, Richard Aloysius	Turners Falls	November	4,	1934	55
1914	McGinity, Joseph Taney	Springfield	December	24,	1934	54
1912	McKelvey, Alexander Dunbar	Ontario, Canada	January	6,	1935	49
1897	Metcalf, Ben Hicks	Ruskin, Florida	March	31,	1935	63
1893	Mitchell, Arthur	Brookline	July	24,	1934	70
1895	†Murphy, Emily Frances	Taunton	November	14,	1934	75
1903	Myers, Solomon	Dorchester	January	18,	1935	62
1895	†Nason, Osman Cleander Baker	Franklin	October	7,	1934	76
1916	Newton, Frank Loomis Sabin	Newtonville	May	30,	1935	77
1911	Nield, William Andrew	Boston	June	12,	1934	59
1915	Nigro, Michele	Medford	August	3,	1934	51
1926	Norton, James Joseph	Hartford, Connecticut	January	19,	1935	50
1921	Ober, Herbert Carroll	Winchester	June	10,	1934	48
1900	O'Brien, Thomas James	Boston	February	5,	1935	62
1896	O'Connor, John Henry	Hyde Park	July	7,	1934	65
1891	Osgood, Gilman	Rockland	September	8,	1934	71
1907	Paddock, Brace Whitman	Pittsfield	May	22,	1935	56
1901	Parcher, George Clarence	Boston	November	29,	1934	63
1908	Pearce, Arthur Cushing	Boston	March	16,	1935	59
1902	†Pearson, Charles Lusby	Newton	September	8,	1934	74
1889	Peirson, Edward Lawrence	Salem	January	18,	1935	72
1907	Pofcher, Elias Henry	Boston	August	28,	1934	53
1927	†Purinton, Herbert Harmon	Somersworth, N. H.	June	7,	1934	69
1896	†Rawson, George Wallace	Amherst	December	27,	1934	73
1903	Richardson, Charles Harper	Pittsfield	May	21,	1935	65
1895	Roberts, Linnaeus Alton	Cambridge	August	23,	1934	66
1890	Robinson, Thomas Johns	Taunton	May	5,	1935	69
1904	Rochette, Edward Charles	Worcester	December	13,	1934	54
1926	‡Rowe, Allan Winter, Ph.D.	Boston	December	6,	1934	55
1892	Rowen, Henry Stanislaus	Brighton	April	29,	1935	66
1932	Russell, Rolfe Spaulding	Greenfield	March	17,	1935	28
1897	Sheehan, Martin David	Winchester	September	4,	1934	63
1890	Simpson, James Edwin	Salem	January	19,	1935	65
1888	Storer, Malcolm	Boston	January	2,	1935	72
1896	Sturgis, Walter Horatio Wakeman	Hull	October	29,	1934	61
1910	Sullivan, Andrew Joseph	Brockton	February	9,	1935	49
1883	†Tallman, Augustus Littlefield	East Boston	May	20,	1934	84
1891	Tracy, Edward Aloysius	Dorchester	January	12,	1935	70
1893	†Trueman, Harmon Silas	Biddeford, Maine	September	2,	1934	76
1886	†Twombly, Edward Lambert	Boston	May	10,	1935	75
1921	Vogel, George Lewis	Boston	March	12,	1935	60
1919	Wagner, Harvey Samuel	Boston	February	9,	1935	57
1892	†Whiting, George Washington Whitney	West Medford	February	14,	1935	70
1904	Whitney, Edward William	Northampton	February	16,	1935	55
1893	Winslow, Edward Smith	So. Harwich	December	28,	1934	68
1919	Wright, Charles Wadsworth	Longmeadow	September	25,	1934	70

†Retired Fellow.
‡Honorary Fellow.

OFFICERS OF THE MASSACHUSETTS MEDICAL SOCIETY FOR 1935-1936

ELECTED BY THE COUNCIL, JUNE 4, 1935

President: Charles E. Mongan, Somerville, 24 Central Street.

Vice-President: Channing Frothingham, Boston. Office, Jamaica Plain, 1153 Centre Street.

Secretary: Alexander S. Begg, West Roxbury. Office, Boston, 8 Fenway.

Treasurer: Charles S. Butler, Boston, 257 Newbury Street.

Orator: Reginald Fitz, Boston, 721 Huntington Avenue.

STANDING COMMITTEES FOR 1935-1936

COMMITTEE OF ARRANGEMENTS

W. R. Morrison, *Chairman;* Horatio Rogers, W. S. Burrage, R. P. Stetson, Augustus Thorndike, Jr.

COMMITTEE ON PUBLICATIONS

R. I. Lee, *Chairman;* Homer Gage, R. B. Osgood, R. M. Smith, F. H. Lahey.

COMMITTEE ON MEMBERSHIP AND FINANCE

D. N. Blakely, *Chairman;* G. C. Caner, J. E. Fish, H. F. Newton, H. Q. Gallupe.

COMMITTEE ON ETHICS AND DISCIPLINE

David Cheever, *Chairman;* W. D. Ruston, S. F. McKeen, A. C. Smith, R. L. DeNormandie.

COMMITTEE ON MEDICAL EDUCATION AND MEDICAL DIPLOMAS

Reginald Fitz, *Chairman;* C. H. Lawrence, C. A. Sparrow, E. S. Calderwood, A. S. Begg.

COMMITTEE ON STATE AND NATIONAL LEGISLATION

C. E. Mongan, *Chairman;* F. E. Jones, A. W. Marsh, A. S. Begg, D. L. Lionberger.

COMMITTEE ON PUBLIC HEALTH

Dwight O'Hara, *Chairman;* G. N. Hoeffel, G. D. Henderson, S. C. Dalrymple, H. L. Lombard.

COMMITTEE ON MALPRACTICE DEFENSE

F. G. Balch, *Chairman;* E. D. Gardner, F. B. Sweet, R. P. Watkins, A. W. Allen.

COMMITTEE ON PERMANENT HOME

R. B. Greenough, *Chairman;* C. G. Mixter, J. M. Birnie, C. S. Butler, E. C. Miller.

SPECIAL COMMITTEES FOR 1935-1936

COMMITTEE ON CANCER

R. B. Greenough, *Chairman;* F. G. Balch, E. M. Daland, P. E. Truesdale, C. C. Simmons.

REPRESENTATIVES TO THE MASSACHUSETTS CENTRAL HEALTH COUNCIL

On Administrative Board
Dwight O'Hara.

District Representatives
Eastern: E. P. Joslin, A. W. Dudley.
Northeastern: M. A. Tighe, F. W. Snow.
Southeastern: W. D. Kinney, C. P. Curley.
Central: G. D. Henderson, E. C. Miller.
Western: H. J. Downey, R. J. Carpenter.

COMMITTEE ON PUBLIC EDUCATION
(A subcommittee of the Standing Committee on Public Health)

G. R. Minot, W. H. Robey, F. J. Cotton, F. C. Irving, R. M. Smith, E. H. Place, C. C. Simmons, J. H. Pratt, H. W. Stevens, J. B. Ayer, H. P. Mosher, R. B. Osgood, F. R. Ober, E. P. Joslin, J. D. Barney, H. L. Lombard.

COMMITTEE ON POSTGRADUATE MEDICAL INSTRUCTION

Frank R. Ober, Boston, *Chairman;* F. Dennette Adams, Boston; Roy Morgan, Westfield; John M. Birnie, Springfield; Harold L. Higgins, Boston; Joseph W. O'Connor, Worcester; Charles W. Blackett, Jr., Newtonville; Dwight O'Hara, Waltham; Reginald Fitz, Boston; Alexander S. Begg, West Roxbury; A. Warren Stearns, Billerica; Robert B. Greenough, Boston; Walter P. Bowers, Clinton; Henry D. Chadwick, Boston; Winfred Overholser, Boston; C. Macfie Campbell, Roxbury; Lincoln Davis, Boston; Leroy E. Parkins, Boston, *Secretary.*

COMMITTEE ON PHYSICAL THERAPY

F. P. Lowry, *Chairman;* R. B. Osgood, G. R. Minot.

COMMITTEE TO CONSIDER THE TYPE OF PERSON TO BE ADMITTED TO THE MASSACHUSETTS MEDICAL SOCIETY AS A FELLOW

R. I. Lee, *Chairman;* A. W. Stearns, H. G. Stetson, P. E. Truesdale, E. R. Leib.

COMMITTEE OF THREE FROM THE MASSACHUSETTS MEDICAL SOCIETY TO CONFER WITH A COMMITTEE OF THREE FROM THE LIBRARY

R. B. Greenough, *Chairman;* P. E. Truesdale, E. C. Miller.

COMMITTEE TO CONSIDER RHEUMATISM AND ALLIED DISEASES IN MASSACHUSETTS

G. R. Minot, *Chairman;* R. B. Osgood, L. T. Swaim, F. R. Ober, L. D. Chapin, G. D. Henderson, H. D. Chadwick, H. L. Lombard, Walter Bauer, *Secretary.*

COMMITTEE ON PUBLIC RELATIONS

One member appointed yearly by each District Medical Society.

The President of the parent society, Charles E. Mongan, is chairman.

Barnstable District Medical Society
Merrill E. Champion, North Harwich.

Berkshire District Medical Society
P. J. Sullivan, Dalton, 46 Curtis Avenue.

Bristol North District Medical Society
Francis H. Dunbar, Mansfield. P. O. address, Boston, 43 Bay State Road.

Bristol South District Medical Society
Edward L. Merritt, Fall River, 130 Rock St.

Essex North District Medical Society
Elmer S. Bagnall, Groveland, 281 Main Street. Secretary.

Essex South District Medical Society
John Joseph Egan, Jr., 52 Pleasant Street, Gloucester.

Franklin District Medical Society
Halbert G. Stetson, Greenfield, 39 Federal Street.

Hampden District Medical Society
Patrick E. Gear, Holyoke, 630 Dwight Street.

Hampshire District Medical Society
Francis E. O'Brien, Haydenville, Hampshire County Sanatorium.

Middlesex East District Medical Society
J. Harper Blaisdell, Winchester. Office Boston, 45 Bay State Road.

Middlesex North District Medical Society
Michael A. Tighe, Lowell, 9 Central Street.

Middlesex South District Medical Society
Charles E. Mongan, 24 Central Street, Somerville.

Norfolk District Medical Society
Walter A. Lane, Milton, 173 School Street.

Norfolk South District Medical Society
William G. Curtis, Wollaston, 10 Grand View Avenue.

Plymouth District Medical Society
Thomas H. McCarthy, Brockton. 142 Main Street.

Suffolk District Medical Society
Channing Frothingham, Boston. Office, Jamaica Plain, 1153 Centre Street.

Worcester District Medical Society
Ernest L. Hunt, Worcester, 28 Pleasant Street.

Worcester North District Medical Society
Harry R. Nye, Leominster, 19 Lancaster Street.

DELEGATES AND ALTERNATES TO THE HOUSE OF DELEGATES OF THE AMERICAN MEDICAL ASSOCIATION

DELEGATES	ALTERNATES
J. M. Birnie, Springfield	W. C. Leary, Springfield
C. E. Mongan, Somerville	L. S. McKittrick, Boston
J. F. Burnham, Lawrence	E. L. Hunt, Worcester
R. H. Miller, Boston	Cadis Phipps, Brookline
E. F. Cody, New Bedford	P. E. Truesdale, Fall River
Reginald Fitz, Boston	G. P. Reynolds, Boston

COUNCILORS—1935-1936

ELECTED BY THE DISTRICT MEDICAL SOCIETIES AT THEIR ANNUAL MEETINGS, APRIL 15 TO MAY 15, 1935

NOTE:—The initials M. N. C. following the name of a Councilor indicate that he is a member of the Nominating Committee. V. P. indicates that a member is a Councilor by virtue of his office as President of a district society and so Vice-President of the general society. C. indicates that he is chairman of a standing committee, Sec. that he is secretary of his District Society.

BARNSTABLE

P. P. Henson, Hyannis, Main St., V. P.
S. M. Beale, Sandwich.
W. D. Kinney, Osterville, M. N. C.
J. I. B. Vail, Hyannis, 155 Main St., Sec.

BERKSHIRE

R. J. Carpenter, North Adams, 85 Main St., V. P.
H. J. Downey, Pittsfield, 184 North St., Sec., M. N. C.
W. T. Frawley, Pittsfield, 184 North St.
G. P. Hunt, Pittsfield, 34 Fenn St.
T. H. Nelligan, Pittsfield, 184 North St.
G. H. Thompson, Pittsfield, 74 North St.

BRISTOL NORTH

L. E. Butler, Taunton, 148 High St., V. P.
W. H. Allen, Mansfield, 70 North Main St., M. N. C.
A. R. Crandell, Taunton, 48 Church Green.
C. B. Kingsbury, Taunton, 63 Prospect St., Sec.
F. V. Murphy, Attleboro, 51 Bank St.

BRISTOL SOUTH

E. L. Merritt, Fall River, 130 Rock St., V. P.
J. A. Barre, Fall River, 1555 Pleasant St.
R. H. Baxter, Marion, 6 South St.
G. W. Blood, Fall River, 82 New Boston Rd.
R. B. Butler, Fall River, 278 North Main St.
E. F. Cody, New Bedford, 105 South Sixth St., M. N. C.
E. D. Gardner, New Bedford, 150 Cottage St.
S. V. Merritt, Fall River, 297 Osborn St.
Charles Shanks, New Bedford, 645 Kempton St., Sec.
I. N. Tilden, Mattapoisett, Barstow St.
C. C. Tripp, New Bedford, 416 County St.
P. E. Truesdale, Fall River, 151 Rock St.

ESSEX NORTH

C. F. Warren, Amesbury, 1 School St., V. P.
E. S. Bagnall, Groveland, 281 Main St., Sec.
R. V. Baketel, Methuen, 7 Hampshire St.
J. F. Burnham, Lawrence, 567 Haverhill St., M. N. C.
H. F. Dearborn, Lawrence, 193 Garden St.
A. P. George, Haverhill, 397 Main St.
T. R. Healy, Newburyport, Office Boston, 370 Marlborough St.
F. W. Snow, Newburyport, 24 Essex St.
L. T. Stokes, Haverhill, 190 Main St.
R. L. Toppan, Newburyport, 148 High St.
W. I. Walker, Andover, 121 Main St.

ESSEX SOUTH

Hanford Carvell, Gloucester, 1038 Washington St., V. P.
N. P. Breed, Lynn, 9 Washington Square.
C. L. Curtis, Salem, 101 Federal St.
C. F. Deering, Danvers, 38 Elm St.
J. F. Donaldson, Salem, 22 Lynde St.
R. E. Foss, Peabody, 125 Main St.
C. L. Hoitt, Lynn, 170 Western Ave., M. N. C.
J. F. Jordan, Peabody, 76 Lynn St.
A. E. Parkhurst, Beverly, 163 Cabot St.
O. S. Pettingill, Middleton, Essex County Sanatorium.
C. H. Phillips, Beverly, 11 Broadway.
W. G. Phippen, Salem, 31 Chestnut St.
R. E. Stone, Beverly, 88 Lothrop Boulevard, Sec.
J. W. Trask, Lynn, 90 Ocean St.

FRANKLIN

H. B. Marble, Shelburne Falls, 63 Bridge St., V.P.
H. M. Kemp, Greenfield, 42 Franklin St., M. N. C.
H. G. Stetson, Greenfield, 39 Federal St., Ex-Pres.
A. H. Wright, Northfield, Main St.
Charles Moline, Sunderland, Office South Deerfield, 120 Main St., Sec.

HAMPDEN

T. S. Bacon, Springfield, 69 Maple St., V. P.
F. H. Allen, Holyoke, 16 Fairfield Ave.
E. P. Bagg, Jr., Holyoke, 207 Elm St.
J. M. Birnie, Springfield, 14 Chestnut St., Ex-Pres.
J. J. Carroll, Holyoke, 172 Chestnut St.
W. A. R. Chapin, Springfield, 121 Chestnut St.
J. L. Chereskin, Longmeadow, Office Springfield, 333 Bridge St.
A. J. Douglas, Westfield, 93 Elm St.
G. L. Gabler, Holyoke, 98 Suffolk St.
P. E. Gear, Holyoke, 630 Dwight St.
Frederic Hagler, Springfield, 20 Maple St.
G. D. Henderson, Holyoke, 312 Maple St.
E. A. Knowlton, Holyoke, 207 Elm St.
M. W. Pearson, Ware, 19 Pleasant St.
A. G. Rice, Springfield, 33 School St.
G. L. Schadt, Springfield, 44 Chestnut St., M. N. C.
H. L. Smith, Springfield, 249 Union St., Sec.
G. L. Steele, Springfield, 20 Maple St.

HAMPSHIRE

L. B. Pond, Easthampton, 115 Main St., V. P.
A. J. Bonneville, Hatfield, 43 Main St.
J. G. Hanson, Northampton, 219 Elm St., M. N. C.
F. E. O'Brien, Haydenville, Hampshire County Sanatorium, Sec.

MIDDLESEX EAST

F. O. West, Woburn, 60 Pleasant St., V. P.
J. H. Blaisdell, Winchester, Office Boston, 5 Bay State Rd.
Richard Dutton, Wakefield, 33 Avon St.
J. H. Fay, Melrose, 49 Lake Ave.
E. M. Halligan, Reading, 3 Salem St.
K. L. Maclachlan, Melrose, 1 Bellevue Ave., Sec.
R. R. Stratton, Melrose, 538 Lynn Fells Parkway, M. N. C.

MIDDLESEX NORTH

E. O. Tabor, Lowell, 18 Shattuck St., V. P.
M. L. Alling, Lowell, 9 Central St.
A. R. Gardner, Lowell, 16 Shattuck St.
J. E. Lamoureux, Lowell, 763 Merrimack St.
G. A. Leahey, Lowell, 128 Merrimack St.
F. R. Mahony, Lowell, 310 Merrimack St., M. N. C.
T. A. Stamas, Lowell, 226 Central St., Sec.
M. A. Tighe, Lowell, 9 Central St.

MIDDLESEX SOUTH

S. H. Remick, Waltham, 775 Trapelo Rd., V. P.
C. F. Atwood, Arlington, 821 Massachusetts Ave.
E. W. Barron, Malden, Office Boston, 20 Ash St.
C. F. K. Bean, West Medford, 51 Harvard Ave.
E. H. Bigelow, Framingham Center, 31 Pleasant St., Ex-Pres.
G. F. H. Bowers, Newton Highlands, 156 Woodward St.
C. O. Chase, Watertown, 6 Patten St.
F. R. Clark, Newtonville, 221 Walnut St.
W. H. Crosby, Brighton, 304 Faneuil St.
A. C. Cummings, Newton, 447 Center St.
D. F. Cummings, Natick, 12 East Central St.
D. E. Currier, Cambridge, 1572 Massachusetts Ave.
J. E. Dodd, Framingham, 141 Franklin St.
D. C. Dow, Cambridge, 1587 Massachusetts Ave.
A. W. Dudley, Cambridge, 1740 Massachusetts Ave., M. N. C.
H. Q. Gallupe, Waltham, 751 Main St.
W. G. Grandison, Charlestown, 65 High St.
W. W. Harrington, Lexington, 1900 Massachusetts Ave.
F. A. Higginbotham, Watertown, 112 Mt. Auburn St.
N. M. Hunter, Hudson, 20 Lincoln St.
C. M. Hutchinson, Cambridge, 47 Garden St.
L. H. Jack, West Newton, 379 Austin St.
A. M. Jackson, Everett, 512 Broadway.

Josephine D. Kable, Marlborough, 42 West Main St.
H. F. Keever, Auburndale, 69 Maple St.
A. A. Levi, Cambridge, Office Boston, 485 Commonwealth Ave., Sec.
F. P. Lowry, Newton, 313 Washington St.
L. W. McGuire, Malden, Office Boston, 395 Commonwealth Ave.
J. A. McLean, West Somerville, 16 Curtis St.
Edward Mellus, Newton, 15 Clements Rd.
C. E. Mongan, Somerville, 24 Central St., Pres.
F. L. Morse, Somerville, 78 Highland Ave.
J. P. Nelligan, Cambridge, 2336 Massachusetts Ave.
D. G. Nutter, Newton Center, 1094 Center St.
E. J. O'Brien, Jr., Brighton, Office Boston, 270 Commonwealth Ave.
Dwight O'Hara, Waltham, Office Boston, 5 Bay State Rd., C.
C. T. Porter, Waltham, Office Boston, 520 Commonwealth Ave.
Ezekiel Pratt, Arlington, 385 Massachusetts Ave.
W. D. Reid, Newton, Office Boston, 510 Commonwealth Ave.
E. F. Sewall, Somerville, 380 Broadway.
F. G. Smith, Somerville, 145 Highland Ave.
C. H. Staples, Malden, 180 Summer St.
H. P. Stevens, Cambridge, 1911 Massachusetts Ave.
H. W. Thayer, Newtonville, 355 Walnut St.
Fresenius Van Nüys, Weston, Boston Post Rd.
H. J. Walcott, Concord, 92 Main St.
W. S. Whittemore, Cambridge, 3 Concord Ave.
Alfred Worcester, Waltham, 314 Bacon St., Ex-Pres.

NORFOLK

L. F. Johnson, Norwood, Office Boston, 15 Bay State Rd., V. P.
H. L. Babcock, Dedham, Office Boston, 99 Bay State Rd.
K. R. Bailey, Jamaica Plain, Office Boston, 483 Beacon St.
Henry Baker, Dorchester, Office Boston, 483 Beacon St.
F. G. Balch, Jamaica Plain, Office Boston, 279 Clarendon St., C.
H. G. Batchelder, Dedham, Office Boston, 510 Commonwealth Ave.
A. S. Begg, West Roxbury, Office Boston, 80 East Concord St., Secretary.
D. D. Berlin, Brookline, Office Boston, 68 Bay State Rd.
D. N. Blakely, Brookline, Office Boston, 87 Milk St., C.
H. K. Boutwell, Brookline, 15 Green St.
F. S. Cruickshank, Brookline, 1236 Beacon St., Sec.
D. G. Eldridge, Dorchester, 15 Monadnock St.
A. A. Fenton, Norwood, 17 Walpole St.
I. A. Finkelstein, Dorchester, 1095 Blue Hill Ave.
J. E. Fish, Canton, Massachusetts State Hospital School.
C. S. Francis, Brookline, 76 High St.
Maurice Gerstein, Roxbury, Office Boston, 483 Beacon St.
Alice M. Gray, Roxbury, 149 Warren St.
W. A. Griffin, Sharon, 28 South Main St.
J. B. Hall, Roxbury, 60 Windsor St.
G. W. Kaan, Sharon, G. P. O.
W. B. Keeler, Roxbury, Office Boston, City Hall Annex.
C. J. Kickham, Brookline, 31 Harvard St.
M. M. Knudson, West Roxbury, 78 Park St.
H. M. Landesman, Roxbury, Office Boston, 463 Commonwealth Ave.
W. A. Lane, Milton, 173 School St.
J. S. H. Leard, West Roxbury, 1895 Center St.
Charles Malone, Jamaica Plain, 46 St. John St.
F. W. Marlow, Jr., Brookline, 1284 Beacon St.
J. S. May, Roxbury, 90 Warren St.
F. P. McCarthy, Milton, Office Boston, 371 Commonwealth Ave.
L. T. McCready, Jamaica Plain, 2 Peter Parley Rd.
S. F. McKeen, Brookline, 96 Dean Rd.
E. F. Murphy, Jamaica Plain, Office Roxbury, 394 Riverway.
T. J. Murphy, Roxbury, 372 Dudley St., M. N. C.
Samuel Nadel, Dorchester, Office Boston, 524 Commonwealth Ave.
Benjamin Parvey, Dorchester, Office Boston, 636 Beacon St.
Cadis Phipps, Brookline, Office Boston, 587 Beacon St.
E. P. Ruggles, Dorchester, Office Boston, 510 Commonwealth Ave.
Victor Safford, Jamaica Plain, 15 Grovenor Rd.
D. D. Scannell, Jamaica Plain, Office Boston, 475 Commonwealth Ave.
A. J. Shadman, Jamaica Plain, 29 Morton St.
J. L. Sullivan, Roxbury, 89 Waverly St.
H. F. R. Watts, Dorchester, 6 Monadnock St.

NORFOLK SOUTH

T. B. Alexander, Scituate Harbor, First Parish Rd., V. P.
C. S. Adams, Wollaston, 62 Brook St.
R. L. Cook, Quincy, 38 Russell Park, Sec.
W. G. Curtis, Wollaston, 10 Grand View Ave.
F. L. Doucett, East Weymouth, 667 Broadway.
G. V. Higgins, Randolph, Warren St.
F. E. Jones, Quincy, 1150 Hancock St.
C. A. Sullivan, South Braintree, 20 Pond St., M. N. C.

PLYMOUTH

W. T. Hanson, Bridgewater, State Farm, V. P.
L. A. Alley, Lakeville, Lakeville State Sanatorium.
W. E. Curtin, Plymouth, 272 Court St.
P. H. Leavitt, Brockton, 129 West Elm St.
T. H. McCarthy, Brockton, 142 Main St., M. N. C.
J. J. McNamara, Brockton, 231 Main St.
C. G. Miles, Brockton, 23 Main St.
G. A. Moore, Brockton, 167 Newbury St., Sec.
A. C. Smith, Brockton, 142 Main St.

SUFFOLK

R. L. DeNormandie, Boston, 355 Marlborough St., V. P.
A. W. Allen, Boston, 264 Beacon St.
J. W. Bartol, Boston, 1 Chestnut St., Ex-Pres.
Horace Binney, Boston, 403 Beacon St.
Gerald Blake, Boston, 311 Beacon St.
W. B. Breed, Boston, 264 Beacon St.
W. J. Brickley, Boston, 524 Commonwealth Ave.
J. E. Briggs, Boston, 477 Beacon St.
C. S. Butler, Boston, 257 Newbury St., Treasurer.
David Cheever, Boston, Peter Bent Brigham Hospital, C.
R. C. Cochrane, Boston, 319 Longwood Ave.
F. H. Colby, Boston, 205 Beacon St.
F. J. Cotton, Boston, 520 Commonwealth Ave.
W. P. Cross, South Boston, 491 Broadway.
Lincoln Davis, Boston, 279 Beacon St., M. N. C.
G. P. Denny, Boston, 66 Commonwealth Ave.
Reginald Fitz, Boston, Peter Bent Brigham Hospital, C.
Channing Frothingham, Boston, Office, Jamaica Plain, Faulkner Hospital, V. P.
Joseph Garland, Boston, 264 Beacon St.

G. L. Gately, East Boston, 624 Bennington St.
R. B. Greenough, Boston, 8 Marlborough St., Ex-Pres., C.
John Homans, Boston, Peter Bent Brigham Hospital.
H. T. Hutchins, Boston, 520 Commonwealth Ave.
E. P. Joslin, Boston, 81 Bay State Rd.
F. H. Lahey, Boston, 605 Commonwealth Ave.
R. I. Lee, Boston, 264 Beacon St., C.
G. A. Leland, Boston, 144 Commonwealth Ave.
C. C. Lund, Boston, 319 Longwood Ave., Sec.
G. B. Magrath, Boston, 274 Boylston St.
J. H. Means, Boston, Massachusetts General Hospital.
W. R. Morrison, Boston, 520 Commonwealth Ave., C.
J. P. O'Hare, Boston, 520 Commonwealth Ave.
A. K. Paine, Boston, 80 Bay State Rd.
F. W. Palfrey, Boston, 10 Post Office Square.
W. S. Parker, Boston, 472 Commonwealth Ave.
W. F. Regan, Chelsea, Office Boston, 19 Bay State Rd.
G. P. Reynolds, Boston, 311 Beacon St.
W. H. Robey, Boston, 202 Commonwealth Ave., Ex-Pres.
G. C. Shattuck, Boston, 240 Longwood Ave.
W. R. Sisson, Boston, 319 Longwood Ave.
Louisa Paine Tingley, Boston, 9 Massachusetts Ave.
G. L. Tobey, Jr., Boston, 270 Commonwealth Ave.
J. R. Torbert, Boston, 252 Marlborough St.
H. P. Towle, Boston, 453 Marlborough St.
Shields Warren, Boston, 195 Pilgrim Rd.
F. A. Washburn, Boston, 150 Bay State Rd.
Conrad Wesselhoeft, Boston, 315 Marlborough St.

WORCESTER

W. F. Lynch, Worcester, 390 Main St., V. P.
J. C. Austin, Spencer, 176 Main St.
W. P. Bowers, Clinton, 264 Chestnut St., Ex-Pres.
L. R. Bragg, Webster, 260 Main St.
F. H. Clapp, North Grafton.
P. H. Cook, Worcester, 27 Elm St.
W. J. Delahanty, Worcester, 5 Trumbull Square.
G. A. Dix, Worcester, 6 Ashland St.
E. B. Emerson, Rutland, Rutland State Sanatorium.
G. E. Emery, Worcester, 340 Main St.
M. F. Fallon, Worcester, 390 Main St.
Homer Gage, Worcester, 8 Chestnut St.
J. J. Goodwin, Clinton, 199 Chestnut St.
David Harrower, Worcester, 13 Elm St., M. N. C.
E. L. Hunt, Worcester, 28 Pleasant St.
E. R. Leib, Worcester, 36 Pleasant St.
A. W. Marsh, Worcester, 690 Main St.
E. C. Miller, Worcester, 27 Elm St., Sec.
J. W. O'Connor, Worcester, 36 Pleasant St.
W. C. Seelye, Worcester, 390 Main St.
E. H. Trowbridge, Worcester, 36 Pleasant St.
F. H. Washburn, Holden, Main St.
R. P. Watkins, Worcester, 332 Main St.
S. B. Woodward, Worcester, 58 Pearl St., Ex-Pres.

WORCESTER NORTH

G. P. Norton, Fitchburg, 24 Prichard St., V. P.
T. R. Donovan, Fitchburg, 42 Fox St.
B. H. Hopkins, Ayer, 24 Washington St.
A. F. Lowell, Gardner, 22 Vernon St.
F. M. McMurray, Fitchburg, 101 Prichard St., Sec.
H. R. Nye, Leominster, 19 Lancaster St., M. N. C.
W. F. Sawyer, Fitchburg, 67 Prichard St.

CENSORS

BARNSTABLE

W. D. Kinney, Osterville, *Supervisor.*
J. H. Higgins, Marstons Mills.
E. E. Hawes, Hyannis.
C. E. Harris, Hyannis.
J. P. Nickerson, West Harwich.

BERKSHIRE

I. S. F. Dodd, Pittsfield, *Supervisor.*
M. S. Eisner, Pittsfield.
J. W. Bunce, North Adams.
G. M. Shipton, Pittsfield.
J. C. Roe, Pittsfield.

BRISTOL NORTH

A. R. Crandell, Taunton, *Supervisor.*
T. F. Clark, Taunton.
T. J. Robinson, Taunton (Deceased).
J. L. Murphy, Taunton.
W. O. Hewitt, Attleboro.

BRISTOL SOUTH

S. V. Merritt, Fall River, *Supervisor.*
D. D. Pratt, New Bedford.
W. F. MacKnight, Fall River.
D. P. O'Brien, New Bedford.
F. M. Howes, New Bedford.

ESSEX NORTH

R. L. Toppan, Newburyport, *Supervisor.*
J. F. Walch, Lawrence.
A. E. Chesley, Lawrence.
W. W. Ferrin, Haverhill.
P. J. Mullen, Amesbury.

ESSEX SOUTH

A. E. Parkhurst, Beverly, *Supervisor.*
S. N. Gardner, Salem.
S. R. Davis, Lynn.
J. J. Hickey, Peabody.
J. J. Egan, Gloucester.

FRANKLIN

H. M. Kemp, Greenfield, *Supervisor.*
A. E. Johnson, Greenfield.
A. H. Ellis, Greenfield.
C. R. Vinal, Turners Falls.
K. H. Rice, South Deerfield.

HAMPDEN

G. L. Gabler, Holyoke, *Supervisor.*
L. D. Chapin, Springfield.
J. A. Seaman, Springfield.
J. E. Dwyer, Springfield.
J. M. Murphy, Palmer.

HAMPSHIRE

A. J. Bonneville, Hatfield, *Supervisor.*
E. W. Brown, Northampton.
J. E. Hayes, Northampton.
M. E. Cooney, Northampton.
C. H. Wheeler, Haydenville.

MIDDLESEX EAST

E. M. Halligan, Reading, *Supervisor.*
C. E. Ordway, Winchester.
C. E. Montague, Wakefield.
F. F. Pike, Melrose.
T. E. Caulfield, Woburn.

MIDDLESEX NORTH

M. L. Alling, Lowell, *Supervisor.*
J. E. Lamoureux, Lowell.
W. F. Ryan, Lowell.
C. S. Baker, Lowell.
J. J. Cassidy, Lowell.

MIDDLESEX SOUTH

H. F. Keever, Newton, *Supervisor.*
T. E. Reilly, Marlborough.
F. W. Gay, Malden.
E. F. Sewall, Somerville.
M. J. Shaughnessey, Framingham.

NORFOLK

E. P. Ruggles, Dorchester, *Supervisor.*
G. W. Winchester, Milton.
H. L. Johnson, West Roxbury.
Maurice Gerstein, Roxbury.
(One to be elected in October.)

NORFOLK SOUTH

C. S. Adams, Quincy, *Supervisor.*
O. H. Howe, Cohasset.
C. J. Lynch, Quincy.
J. H. Cook, Quincy.
D. L. Belding, Hingham.

PLYMOUTH

J. J. McNamara, Brockton, *Supervisor.*
L. A. Baker, Middleborough.
A. W. Carr, Bridgewater.
R. B. Rand, North Abington.
D. B. Tuholski, Brockton.

SUFFOLK

G. L. Tobey, Jr., Boston, *Supervisor.*
Gerald Blake, Boston.
W. B. Breed, Boston.
F. H. Colby, Boston.
Shields Warren, Boston.

WORCESTER

G. A. Dix, Worcester, *Supervisor.*
J. J. Dumphy, Worcester.
L. M. Felton, Worcester.
T. F. O'Brien, Worcester.
H. V. Williams, Whitinsville.

WORCESTER NORTH

T. R. Donovan, Fitchburg, *Supervisor.*
E. A. Adams, Fitchburg.
A. A. Wheeler, Leominster.
F. H. Thompson, Jr., Fitchburg.
J. G. Simmons, Fitchburg.

PRESIDENTS OF DISTRICT MEDICAL SOCIETIES

VICE-PRESIDENTS (*Ex-Officiis*)

Arranged according to seniority of fellowship in the Massachusetts Medical Society.

T. S. Bacon, Springfield.
G. P. Norton, Fitchburg.
T. B. Alexander, Scituate Harbor.
R. L. DeNormandie, Boston.
W. T. Hanson, Bridgewater.
L. B. Pond, Easthampton.
F. O. West, Woburn.
W. F. Lynch, Worcester.
Hanford Carvell, Gloucester.
E. O. Tabor, Lowell
R. J. Carpenter, North Adams.
C. F. Warren, Amesbury.
E. L. Merritt, Fall River.
P. P. Henson, Hyannis.
S. H. Remick, Waltham.
L. F. Johnson, Norwood.
H. B. Marble, Shelburne Falls.
L. E. Butler, Taunton.

COMMISSIONERS OF TRIALS

BARNSTABLE—F. O. Cass, Provincetown.
BERKSHIRE—I. S. F. Dodd, Pittsfield.
BRISTOL NORTH—R. P. Kent, Attleborough.
BRISTOL SOUTH—A. C. Lewis, Fall River.
ESSEX NORTH—R. C. Hurd, Newburyport.
ESSEX SOUTH—O. C. Blair, Lynn.
FRANKLIN—J. A. Mather, Greenfield.
HAMPDEN—J. M. Birnie, Springfield.
HAMPSHIRE—E. H. Copeland, Northampton.
MIDDLESEX EAST—C. E. Ordway, Winchester.
MIDDLESEX NORTH—H. W. Jewett, Lowell.
MIDDLESEX SOUTH—E. P. Stickney, Arlington.
NORFOLK—H. F. R. Watts, Dorchester.
NORFOLK SOUTH—N. S. Hunting, Quincy.
PLYMOUTH—F. F. Weiner, Brockton.
SUFFOLK—Channing Frothingham, Boston.
WORCESTER—W. P. Bowers, Clinton.
WORCESTER NORTH—H. R. Nye, Leominster.

OFFICERS OF THE DISTRICT MEDICAL SOCIETIES

ELECTED BY THE DISTRICT MEDICAL SOCIETIES AT THEIR ANNUAL MEETINGS, BETWEEN APRIL 15, AND MAY 15, 1935.

(The street address may be obtained from the Annual Directory.)

BARNSTABLE—*President*, P. P. Henson, Hyannis; *Vice-President*, M. E. Champion, North Harwich; *Secretary*, J. I. B. Vail, Hyannis; *Treasurer*, H. B. Hart, Yarmouthport; *Librarian*, E. E. Hawes, Hyannis.

BERKSHIRE—*President*, R. J. Carpenter, North Adams; *Vice-President*, W. T. Frawley, Pittsfield; *Secretary*, H. J. Downey, Pittsfield; *Treasurer*, C. T. Leslie, Pittsfield.

BRISTOL NORTH—*President*, L. E. Butler, Taunton; *Vice-President*, H. L. Rich, Attleboro; *Secretary*, C. B. Kingsbury, Taunton; *Treasurer*, J. V. Chatigny, Taunton.

BRISTOL SOUTH—*President*, E. L. Merritt, Fall River; *Vice-President*, J. M. Bonnar, New Bedford; *Secretary* and *Treasurer*, Charles Shanks, New Bedford.

ESSEX NORTH—*President*, C. F. Warren, Amesbury; *Vice-President*, E. P. Laskey, Haverhill; *Secretary* and *Treasurer*, E. S. Bagnall, Groveland; *Auditor*, A. M. Hubbell, Haverhill.

ESSEX SOUTH—*President*, Hanford Carvell, Gloucester; *Vice-President*, J. F. Bradley, Peabody; *Secretary*, R. E. Stone, Beverly; *Treasurer*, Andrew Nichols, III, Danvers.

FRANKLIN—*President*, H. B. Marble, Shelburne Falls; *Vice-President*, W. J. Pelletier, Turners Falls; *Secretary* and *Treasurer*, Charles Moline, Sunderland.

HAMPDEN—*President*, T. S. Bacon, Springfield; *Vice-President*, P. E. Gear, Holyoke; *Secretary* and *Treasurer*, H. L. Smith, Springfield.

HAMPSHIRE—*President*, L. B. Pond, Easthampton; *Vice-President*, Ward Young, Northampton; *Secretary* and *Treasurer*, F. E. O'Brien, Haydenville; *Librarian*, Jane B. Armstrong, Northampton.

MIDDLESEX EAST—*President*, F. O. West, Woburn; *Vice-President*, J. H. Kerrigan, Stoneham; *Secretary*, K. L. Maclachlan, Melrose; *Treasurer*, Richard Dutton, Wakefield; *Librarian*, J. M. Wilcox, Woburn.

MIDDLESEX NORTH—*President*, E. O. Tabor, Lowell; *Vice-President*, F. P. Murphy, Lowell; *Secretary*, T. A. Stamas, Lowell; *Treasurer*, M. D. Bryant, Lowell; *Librarian*, P. J. Meehan, Lowell.

MIDDLESEX SOUTH—*President*, S. H. Remick, Waltham; *Vice-President*, F. R. Jouett, Cambridge; *Secretary*, A. A. Levi, Cambridge; *Treasurer*, Edward Mellus, Newton; *Librarian*, J. M. Baty, Framingham.

NORFOLK—*President*, L. F. Johnson, Norwood; *Vice-President*, Maurice Gerstein, Roxbury; *Secretary*, F. S. Cruickshank, Brookline; *Treasurer*, G. W. Kaan, Sharon.

NORFOLK SOUTH—*President*, T. B. Alexander, Scituate; *Vice-President*, N. R. Pillsbury, Braintree; *Secretary*, R. L. Cook, Quincy; *Treasurer*, F. W. Crawford, Holbrook; *Librarian*, R. L. Cook, Quincy.

PLYMOUTH—*President*, W. T. Hanson, Bridgewater; *Vice-President*, Charles Hammond, Hanover; *Secretary*, G. A. Moore, Brockton; *Treasurer*, A. C. Smith, Brockton; *Librarian*, J. H. Weller, Bridgewater.

SUFFOLK—*President*, R. L. DeNormandie, Boston; *Vice-President*, Conrad Wesselhoeft, Boston; *Secretary*, C. C. Lund, Boston; *Treasurer*, John Rock, Boston.

WORCESTER—*President*, W. F. Lynch, Worcester; *Vice-President*, R. J. Ward, Worcester; *Secretary*, E. C. Miller, Worcester; *Treasurer*, E. P. Disbrow, Worcester.

WORCESTER NORTH—*President*, G. P. Norton, Fitchburg; *Vice-President*, H. W. Ellam,* Gardner; *Secretary*, F. M. McMurray, Fitchburg; *Treasurer*, F. H. Thompson, Jr., Fitchburg.

*Deceased.

HYPERINSULINISM*

BY J. ARTHUR BARNES, M.D.,† AND EUGENE L. RICHMOND, M.D.†

HYPERINSULINISM, the syndrome first described by Harris in 1923, is the condition in which the pancreas produces so much insulin that hypoglycemia and its attending symptoms occur. While the disease entity is probably not new, the mechanism was not recognized or understood until the isolation of insulin and its use to combat diabetes.

Spontaneous hyperinsulinism is to be distinguished from spontaneous hypoglycemia which is merely a descriptive term indicating the lowering of the blood sugar which may be induced by a variety of causes. Hyperinsulinism is considered to be due to overactivity of the islands of Langerhans and this overactivity may be due to diffuse hyperplasia or to adenoma formation.

The clinical picture of hypoglycemia is familiar to all who have had experience with the use of insulin. It is only recently that disturbances of the pancreas other than hypofunction of the islands of Langerhans with its anatomic changes of atrophy and production of diabetes have been reported. There have been increasing observations of spontaneous hyperfunction associated with hypertrophy and adenoma of the pancreas with the production of hyperinsulinism. It is apparent that the sequence, hyperfunction, hyperplasia, tumor formation against hypofunction, hypoplasia, aplasia destruction is manifest in the pancreas as with the earlier studies of the thyroid and the pituitary.

In a résumé of the cases reported it is stated that most patients presented a history of gradual onset with symptoms varying from hunger, weakness, dizziness and a feeling of apprehension to that of momentary loss of consciousness with associated automatic movements lasting from a few seconds to several moments. These symptoms all followed a long starvation period at first and were especially common before breakfast. They were likewise often seen when severe muscular exercise directly preceded a meal. They usually disappeared or could be aborted by the ingestion of food. There was a tendency for the attacks to become more frequent and more severe until, by the time the patient was seen, convulsive attacks associated with coma were common. Another finding which was marked in several patients was a definite mental deterioration. A striking feature in the typical history is that the patient usually notices the association between attacks and hunger. The symptoms of the late cases are identical with those of insulin shock; weakness, headache, nervousness, anxiety, fatigability, coldness, numbness of the extremities, epigastric distress, tremor, profuse perspiration and visual disturbances. In short the symptoms are so protean and indefinite that the busy practitioner is too liable to dismiss the case as one of hysteria or nervousness, and prescribe one of the many mild sedatives and is quite surprised that these are of no benefit and the patient progresses to the typical attacks of actual coma. Those cases seen with convulsions in which the attack is preceded by typical premonitory symptoms of dizziness, chilliness, profuse perspiration, a craving for sweets, an involuntary cry or a fall, with increasing drowsiness followed by coma with muscular twitching, at first limited to the hands or face followed by generalized rigidity and salivation are often mistakenly diagnosed as epilepsy or the convulsions of a brain tumor. In one series the diagnosis of idiopathic epilepsy was made fourteen times in a series of sixty-five reviewed cases of spontaneous hypoglycemia. Because of this close clinical relationship it is strongly recommended that all epileptics have blood sugar studies as there seems to be a small number whose convulsions are secondary to hypoglycemia.

Physical examination usually reveals nothing

*Read before the Worcester District Medical Society at St. Vincent Hospital, December 12, 1934.

†Barnes, J. Arthur—Attending Surgeon, St. Vincent Hospital, Worcester. Richmond, Eugene L.—Assistant Attending Surgeon, St. Vincent Hospital, Worcester. For records and addresses of authors see "This Week's Issue," page 240.

of any significance. There is a tendency for the patients to be overweight due to the excessive intake of carbohydrate. Blood pressure readings are commensurate with the age of the patient. Basal metabolic readings are not significant although in many cases this was not done due to the danger of a long fast.

The dextrose tolerance curve varies. In the functional group the elevation of sugar level is small, rarely an increase of so much as fifty per cent in one hour and a drop to the prefasting level or below in three hours. Yet there have been five reported exceptions to this observation. In the majority of cases with adenoma the elevation is in excess of normal and the fall to the low level is delayed. However, there have not been enough observations to use this test to differentiate the three types of pathology in the pancreas, hyperplasia, adenoma and cancer.

Epinephrin is supposed to exert an hyperglycemic effect by the liberation of sugar from the glycogen in the liver. In normal individuals the injection of 0.6 cc. of a 1:1000 solution of adrenalin causes an increase in blood sugar of thirty-five to forty-five mgm. within forty-five to sixty minutes not returning to the normal level for two hours. Theoretically one would expect that adrenalin would be efficacious in raising the blood sugar level in cases of hypoglycemia of pancreatic origin and not in cases of hepatic origin. In the reported cases the elevation is more prompt than in normal cases. The rise is almost as high and there is a more rapid drop to the pre-injection level. Because of this fact the administration of adrenalin in an attack is very beneficial but it must be followed at once with ingestion of glucose to maintain the elevation.

Pituitrin, although it has some antagonistic effect on the action of insulin, has not proved of value in warding off attacks.

Thyroid extract is somewhat more potent. If enough extract is given to maintain a metabolic elevation of twenty per cent, the tendency to attacks will be checked. The disadvantages of this form of therapy are obvious.

An accepted classification of the causes of hypoglycemia is as follows:

(1) Excess of insulin as a result of therapeutic injections of insulin, of tumors and hyperplasia of the pancreas or of functional hyperinsulinism.

(2) Lack of opposing secretions may result from diseases of the suprarenal glands, tumors of the anterior or posterior lobe of the pituitary or from myxedema.

(3) Lack of glycogen.

This lack may result from destruction of reservoirs, disease of the liver or wasting of muscles, from abnormal secretion of sugar in starvation and intoxications from phosphorus or arsphenamine poisons.

(4) Interference with regulating center. This may result from disease affecting the pons or from overaction of the vagus nerves.

Accepting the facts that studies of the blood sugar in the fasting state and for dextrose tolerance curves and that the therapeutic use of dextrose reasonably establishes the occurrence of hyperinsulinism, the problem of determining the nature of the pancreatic pathology presents a great many difficulties. A localized malignant growth may be found. The impossibility of resolving the diagnostic uncertainty of the underlying pancreatic pathological changes and of thereby determining the operative procedure make the decision for operation quite difficult as only exploratory intervention can be done and the prognosis is uncertain. Medical treatment by excessive feeding, especially carbohydrates, is only a temporary procedure and in many cases, as with the present reported one, will not relieve the attacks. Then, too, there is always the possibility of increased hyperfunction due to the stimulation of forced glucose feeding which will speed up this progressive disease entity.

From the few papers concerned with the intensive study of these cases it is apparent that there is no striking difference between those with an adenoma and those with no demonstrable pancreatic lesion. For this reason it is suggested that all patients be given a trial at medical treatment for a suitable interval. There is a very grave note of warning suggested in the report of one author (Wilder) who found two cases of carcinoma with metastasis in fourteen cases. This ever-present danger coupled with the fact that a medical régime has not been accepted by all men as being satisfactory, many recommending frequent high carbohydrate feedings, others high fat, low carbohydrate, would seem to indicate that one cannot dally too long. The reasoning of those advocating a high fat diet seems to be more sound as repeated carbohydrate feeding would tend to cause excessive production of insulin by a gland already hyperfunctioning. This is well borne out by the clinical history of those patients who early recognize the value of food especially quickly assimilated carbohydrate and their rapidly progressive decline.

Another (Rynearson) states that it would seem advisable to subject all patients with this syndrome to abdominal exploration since there is no method of diagnosing it preoperatively. He bases his premise on the belief that all these tumor cells are neoplastic with some resemblance to island cells and producing insulin or some related substance. He further states that so many carcinomata give no warning signs; yet here is a type that shrieks for it.

Experience with surgical treatment to date, however, has not been entirely satisfactory. When an adenoma has been found and removed a cure has resulted, but if a tumor has

not been found and if resection of the pancreas has been attempted, the benefit has been slight. However, many authors feel that the gland can be resected with safety. The probable cause of the hyperplastic cases is that not enough gland substance has been removed, again paralleling early thyroid surgery.

We are indebted to Dr. M. Gene Black for a complete medical and laboratory investigation during the eighteen months previous to our investigations when the patient consulted her at irregular intervals. During this period the patient had consulted numerous physicians who had told her that her symptoms were due to a "nervous condition". Medication for this nebulous diagnosis had given no relief.

Report of a case:

K. P. B., single female, aged thirty-five.

Past history: uneventful but for a D. & C. and cauterization of the cervix for pelvic pain and vaginal discharge three years ago. Blood count, blood pressure and urine were negative at that time. Blood chemistry studies were not recorded.

The exact beginning of the present illness was difficult to determine because of the vagueness of the symptoms. Eighteen months ago she sought medical relief for a fall. Blood pressure, blood count and urine were negative at this time. The patient was extremely nervous and emotionally unstable, all manipulations causing extreme apprehension. In retrospect this was probably the beginning of the present syndrome.

For approximately one year the patient has been suffering from fainting and sinking spells lasting from ten to fifteen minutes during which there was loss of consciousness with clonic and tonic contractions. Recovery was always spontaneous leaving the patient fatigued and exhausted. There was no conscious association with the taking of food to allay or prevent the attacks although the patient stated that she has always had a craving for sweets, has been consuming candy in larger quantities lately and always has some with her. In spite of the increased carbohydrate intake there has been no increase in weight. The attacks have had a definite periodicity occurring either at ten A.M. or three P.M. with luncheon at twelve noon. They have been of increasing frequency and duration. Examination at this time revealed nothing significant. There was slight tenderness in the L.U.Q. In addition to the fainting spells the patient complained of weakness, fatigue, coldness, irritability and insomnia. There was definite emotional instability as indicated by frequent crying spells and marked apprehension during the examination. Basal metabolism was +3 and +4 in repeated tests. Previous basal metabolism tests one year ago revealed +9, —3.5, —7.5.

The patient was placed upon a high carbohydrate diet with marked immediate relief of symptoms. Subsequent blood sugar determinations were as follows:

Fasting	6:00 A.M.	30	mgm.	per	100	cc.	blood
	7:30 A.M.	83	"	"	"	"	"
	4:30 P.M.	90	"	"	"	"	"
	10:00 P.M.	68	"	"	"	"	"
	11:00 P.M.	41	"	"	"	"	"

on different days without controlled high carbohydrate diet.

On admission to the hospital, blood sugar fasting specimen was sixty mgm. After the ingestion of 100 Gm. of glucose, the readings of the glucose tolerance test were the following: one-half hour, eighty mgm.; one hour, seventy-eight mgm.; two hours, eighty-four mgm.; three hours, ninety-four mgm.; four hours, eighty mgm. At no time was there any overflow of sugar into the urine.

During the test the patient had two typical attacks during which there was generalized rigidity of the entire body, eyes fixed, face and mouth rigid. The B.P. remained normal and there was no change in the pulse rate. The patient did not respond to questions or pressure over the supraorbital nerves. The first attack was of ten minutes' duration, the second of fifteen. Recovery was spontaneous followed by lassitude and prostration and alternating periods of crying and forced gaiety. This type of curve as contrasted with the reported observations would indicate the pathology in the pancreas to be of the hyperplastic type. Yet of those reported, five were marked exceptions to this and in one of them carcinoma was found at postmortem.

She was placed upon a high carbohydrate diet and the following morning the blood sugar was 115 mgm. One-half hour after the injection of 0.5 cc. 1:1000 adrenalin the blood sugar was 150 mgm. which persisted for one hour. Inasmuch as epinephrin is supposed to exert its hyperglycemic effect by the liberation of sugar from the glycogen in the liver, it would indicate in this case that the patient had adequate stores in the liver and that it was readily available and that the hyperinsulinism was not due to liver pathology.

Basal metabolism tests ruled out any thyroid disorder as the basic cause and there were no stigmata to indicate pituitary dysfunction.

Because of the increase in symptoms and the progressive nature of the disease as seen in many reported cases diagnosed only at the autopsy table, a loss of fifteen lbs. in six weeks and bearing in mind the warnings issued by authors reporting well-developed malignant growths, exploratory intervention was decided upon even though a clean-cut differentiation of hyperplasia, adenoma and cancer could not be predetermined.

At operation adequate exposure was obtained through the gastrocolic omentum. Thorough palpation of the entire gland failed to reveal any discrete adenomata. The major portion of the gland was softer and paler than in the normal organ. At the extreme tail of the pancreas adjacent to the spleen there was a shotty like feel to the gland. Approximately two inches of the tail was then resected.

Following the operation and four hours after the intravenous injection of sixty Gm. of glucose the blood sugar was 333 mgm. with 0.5 per cent in the urine. Two days later the sugar was 178 mgm. on forced carbohydrate feeding. The persistence of sugar in the blood was an encouraging sign that the rapid formation of insulin had been checked.

Three days postoperatively the patient developed a diffuse bronchopneumonia followed by acute dilation of the stomach and died. Autopsy permission was not obtained.

Pathologic report:

Section through the pancreas shows an increase in the interstitial fibrous tissue and an irregular infiltration of lymphocytes about the islands. There were no marked abnormalities histologically involving the islands themselves of the sections studied. From these sections it would appear that the hypersecretion was due to overactivity of normal appearing epithelium.

There is definite need for more elaborate studies of this clinical entity. This paper is intended primarily to bring the syndrome before the profession so that earlier diagnosis of this condition can be made. Unquestionably it is

much more frequent than the literature would indicate, and a plea is made for a more thorough investigation of those obscure cases that we all too readily dismiss as being functional in origin.

REFERENCES

1. Cairns, R. McD., and Tanner, S. E.: Adenoma of islets of Langerhans associated with hypoglycaemia. Brit. M. J. 1:8 (Jan. 7) 1933.
2. Carr, Archie D.; Parker, Robert; Grove, Edward; Fisher, A. O., and Larimore, J. W.: Hyperinsulism from B-cell adenoma of the pancreas; operation and cure. J. A. M. A. 96:1363 (April 25) 1931.
3. Frank, Heinz: Fatal hypoglycemia in adenoma of the pancreas. Deutsche Arch. f. klin. Med. 171:175, 1931.
4. Graham, E. A., and Womack, N. A.: The application of surgery to the hypoglycaemic state due to islet tumors of the pancreas and to other conditions. Surg. Gynec. & Obst. 56:728, 1933.
5. Harris, Seale: Hyperinsulinism and dysinsulinism (insulinogenic hypoglycemia) with a chronological review of cases reported in the United States and Canada. Internat. Clin. Forty-Second Series. 1:9 (March) 1932.
6. Harris, Seale: Hyperinsulinism and dysinsulinism. J. A. M. A. 83:729 (Sept. 6) 1924.
7. Howland, Goldwin; Campbell, Walter R.; Maltby, Ernest J., and Robinson, W. L.: Dysinsulinism; convulsions and coma due to islet cell tumor of the pancreas with operation and cure. J. A. M. A. 93:674 (Aug. 31) 1929.
8. Jacobs, Sydney: Hyperinsulinism; with report of a case. New Orleans M. & S. J. 86:724 (May) 1934.
9. Rathery, F., and Sigwald, J.: Fatal hypoglycemia during the course of treatment with insulin. Gaz. méd. de France. P. 384 (June 1) 1932.
10. Rynearson, E. H., and Moersch, F. P.: Neurologic manifestations of hyperinsulinism and other hypoglycemic states. J. A. M. A. 103:1196 (Oct. 20) 1934.
11. Tedstrom, Milo K.: Hypoglycemia and hyperinsulinism. Ann. Int. Med. 7:1013 (Feb.) 1933-34.
12. Terbrüggen, A.: Diabetes mellitus, regeneration of the islands of Langerhans and adenoma of the islands of Langerhans, pancreatic hyper- and hypoglycemia. Munchen. med. Wchnschr. 80²:1705 (Oct. 27) 1933.
13. Thalhimer, W., and Murphy, Francis D.: Carcinoma of the islands of the pancreas, hyperinsulinism and hypoglycemia. J. A. M. A. 91:88 (July 14) 1928.
14. Weil, Clarence K.: Functional hyperinsulinism; epileptiform convulsions accompanying spontaneous hypoglycemia. Internat. Clin. 4:33 (Dec.) 1932.
15. Wilder, Russell M.: Hyperinsulinism. Internat. Clin. 2:1 (June) 1933.
16. Womack, Nathan A.: Hypoglycemia due to adenoma of the islets of Langerhans. South. M. J. 27:135 (Feb.) 1934.

BRAIN ABSCESS AS A COMPLICATION OF SEPTIC PLEUROPULMONARY DISEASE*

BY D. A. NICKERSON, M.D.†

INTRODUCTION

THE unexpected, sudden appearance of symptoms of suppurative disease of the brain as a complication of septic pleuropulmonary disease has long been recognized. Why the brain is attacked more frequently than the other organs has always been a puzzling question and the relative rarity of metastatic abscesses in acute septic conditions, such as endocarditis, makes it even more difficult to understand. Many theories have been advanced, but none have been entirely acceptable. Schorstein[1] felt that in acute septic diseases, the brain has a greater resistance, while in chronic pulmonary disease, it is not so fully protected as other organs by locally formed antitoxic agents. Eagleton[2] asserts that the cerebral metastases from the lungs are primarily of venous or thrombotic origin as contrasted with abscesses of embolic or arterial origin in acute septic cases. He feels that pulmonary disease favors the thrombotic process in the cerebral veins, and substantiates his views by Arnold's[3] experiment. The latter injected foreign material into the larger neck veins and by producing a positive intrathoracic pressure found the material in the cerebral veins. This was explained as a retrograde venous phenomenon, but the relatively minor venous return by the azygos veins refutes this view. Gardner[4] felt that long-continued coughing, by producing a positive intrathoracic pressure, lowers the cardiac output temporarily, and a consequent transient ischemia of areas of the brain develops which tends to lower cerebral resistance. Proof of this momentary ischemia is seen in the temporary unconsciousness occasionally noted in vital capacity tests. Parker[5] questions whether it is possible that the tissues of the lung and brain have something in common whereby brain tissue acts as a favorable soil for metastases of suppurative or carcinomatous processes of the lungs.

Following Schorstein's paper, cases of septic pleuropulmonary disease with subsequent brain abscesses have been divided into several types dependent upon the pathological lesion in the lung; chiefly, lung abscess, empyema and bronchiectasis. Such a classification is necessarily arbitrary since the pathological boundaries are not always clear and many cases combine several types of lesions. Many authors do not separate their cases properly, so that figures regarding the incidence of the various processes in the lung and metastatic cerebral abscesses are subject to criticism.

MATERIAL

This report is based upon a study of sixty cases of brain abscess and 538 cases of septic pleuropulmonary disease occurring in a series of 10,502 autopsies performed in this laboratory over a period of thirty-nine years (1896-1934). This has been done in an attempt to establish the incidence and location of brain abscesses associated with septic disease of the lung and pleura.

Of the sixty cases of brain abscess, twenty-seven (45 per cent) were secondary to otogenic infections, a figure in accord with most writers. Twelve cases (20 per cent) were secondary to disease in the lungs; six cases (10 per cent) were associated with septicemia; five cases (8.4 per

*From the Mallory Institute of Pathology, Boston City Hospital.

†Nickerson, D. A.—Assistant in Pathology, Mallory Institute of Pathology, Boston City Hospital. For record and address of author see "This Week's Issue," page 240.

cent) occurred with acute endocarditis; two cases (3.3 per cent) were consequent to septic disease of the orbit and eight cases (10 per cent) were sequelae of various other diseases.

In correlating these figures with the analysis of 538 cases of pulmonary disease only those cases where examination of the head was done, of which there were 180 cases, are included in this study. Seventy-four cases of lung abscess showed eleven brain abscesses, a percentage of 14.7. Sixty-six cases of empyema showed but one cerebral abscess, a percentage of 1.5. Forty cases of bronchiectasis showed no cerebral metastases.

DISCUSSION

Table 1 gives the essential details of the twelve cases of cerebral abscess which occurred as sequelae of suppurative disease of the lung and pleura. Of these, seven cases (58 per cent) followed single lung abscess; four cases (33 per cent) were associated with multiple lung abscesses. One case was secondary to empyema. These figures are in direct contrast to Schorstein, who collected sixty-nine cases, nineteen of which were his own. He found bronchiectasis present in thirty-eight cases (55 per cent), empyema in fifteen (23 per cent), gangrene of the lung in six (9 per cent) and the remaining ten were evenly divided with abscess of the lung present in only two cases. The figures are in keeping, however, with more recent reports. Cohen[6] in reporting nineteen cases, found that fourteen (73 per cent) were associated with lung abscess of which ten were single and four were multiple. Four of his cases (21 per cent) were secondary to empyema, and one case (5 per cent) followed bronchiectasis. No cases associated with pulmonary tuberculosis were found in this study, which is in accord with most authors, although Parker[5] reports one case, and also cites a case secondary to pulmonary actinomycosis.

In this present series, eight of the cerebral abscesses (66 per cent) were single and four multiple. This incidence of solitary abscesses is higher than that usually quoted. Schorstein[1] found that thirty-two (62 per cent) were multiple, and nineteen were single. Eagleton[2] in a review of the literature estimated that 45 per cent were single. Parker[5] found that his fourteen cases were evenly divided, seven being single and seven multiple.

There appears to be a slight predominance of localization in the parietal lobe in this group; four of the nine solitary abscesses being found in this area. There was but one abscess found in the frontal lobe, which indicates that localization here is a rarity in contrast to the commonly accepted belief. Of interest, is one case involving the cerebellum as this is an unusual site, Groth[7] stating he had never seen one.

As to the side of the brain involved, no direct relationship existed between it and the side on which the primary lung pathology was found.

TABLE 1

			Brain Abscess		*Lung Abscess*	
No.	Sex	Age	Type	Location	Type	Location
97-378	Male	20	Single	Left Frontal	Single	Right Apex
99-69	Male	?	Double	Right Occipital Right Cerebellum	Single	Right Apex
01-29	Female	35	Single	Right Parieto-Occipital	Single	Left Base
02-49	Female	42	Multiple	Throughout	Multiple	Throughout
02-179	Female	58	Single	Left Basal Ganglia	Empyema	Left
06-151	Female	9	Single	Left Parietal	Single	Left Base
24-122	Female	26	Single	Right Parietal	Multiple	Right Base
24-173	Male	41	Single	Cerebellum (Uvula)	Single	Left Apex
26-154	Male	36	Multiple	Throughout	Bilateral	Both Bases
28-83	Male	27	Single	Left Parietal	Multiple	Left Base
32-465	Male	50	Multiple	Throughout	Single	Right Apex
33-7	Male	32	Single	Left Parietal	Multiple	Right Lung

No age group was preponderantly involved, the cases being quite evenly divided between the first to sixth decade. There was, however, a distinct predominance of the male sex by a ratio of 2:1.

No definite conclusions could be drawn from the bacteriological studies. The otogenic cases showed a predominance of hemolytic streptococci. Those secondary to disease of the lung showed a wide variety of organisms. The septicemia cases were chiefly hemolytic streptococci; one unusual case, however, was found to have B. mallei in the heart's blood and brain. Of the acute endocarditis cases, two showed hemolytic streptococci, and two staphylococcus aureus in the heart's blood.

SUMMARY

1. Sixty cases of brain abscess and 528 cases of septic pleuropulmonary disease have been studied in an attempt to establish the incidence and location of cerebral metastases in suppurative disease of the lung and pleura.

2. Twelve cases of metastatic cerebral abscesses are presented in detail.

3. Of the etiological factors in brain abscess,

suppurative disease of the lung and pleura was responsible for twelve of the sixty cases, being second only to otogenic infections as a cause.

4. Abscess of the lung was the most common antecedent pulmonary disease, being found in eleven of the twelve cases. Of these, the lung abscess was solitary in seven cases; multiple in four.

5. Cerebral metastases occurred in eleven of seventy-four cases of pulmonary abscess, an incidence of 14.7 per cent.

6. Only one brain abscess was found in a series of sixty-six cases of empyema, an incidence of 1.5 per cent.

7. No metastatic abscesses were found in forty cases of bronchiectasis.

8. Eight of the cerebral abscesses were single; four were multiple.

9. The parietal lobe appears to be a site of predilection, four of the seven solitary abscesses occurring here.

10. The literature is briefly reviewed.

REFERENCES

1. Schorstein, G. I.: Abscess of the brain in association with pulmonary disease. Lancet 2:843 (Sept. 18) 1909.
2. Eagleton, W. P.: Brain Abscess. New York: Macmillan Co. P. 68, 1922.
3. Arnold, J.: Ueber rückläufigen Transport. Arch. f. path. Anat. 124:385, 1891.
4. Gardner, W. J.: Metastatic abscesses of the brain of pulmonary origin: report of 2 cases. Arch. Neurol. & Psychiat. 19:904, 1928.
5. Parker, H. L.: Metastatic abscesses of the brain. Am. J. M. Sc. 180:699, 1930.
6. Cohen, I.: Cerebral complications of putrid pleuropulmonary suppuration. Arch. Neurol. & Psychiat. 32:174, 1934.
7. Groth: Quoted by Eagleton.

EXPERIENCES IN IONIZATION OF THE NASAL MUCOUS MEMBRANE

BY HAROLD G. TOBEY, M.D.*

THE theory of ionization of mucous membranes by metallic solutions aided by an electrical current has been applied for more than a decade, but has not been widely practiced. A new impetus has been given to this therapeutic measure through the work of Dr. H. L. Warwick who has investigated the results to be obtained from various metallic solutions and their combinations, and has developed a means by which a definitely controlled smooth direct current of electricity may be passed through the structures to be treated without harm to the tissues or discomfort to the subject.

The theory, briefly stated, is that a metallic solution placed in contact with the nasal mucous membrane results in the precipitation of the proteins of the superficial cells, but when a mild direct current is passed through the tissues the metallic ions penetrate more deeply and precipitate more of the proteins of the cells. Great care is taken that the strength of the current does not reach the point of coagulation of the tissues.

Warwick, after considerable experimentation, has adopted a solution consisting of sulphate of zinc 85 per cent, tin 10 per cent and cadmium 5 per cent, as best suited for the treatment. An alloy of these three metals, in the same proportions, is used for the anode.

The method of treatment consists in thoroughly anesthetizing the entire mucous membrane, following which, the nasal cavities are thoroughly packed with cotton strip saturated with the metallic solution and the anode introduced. The cathode is then affixed to the arm of the same side. The efficiency of the packing is indicated by a resistance ohmmeter which is incorporated in the instrument. The resistance should be from 2500 to 3000 ohms for a satisfactory result. If this resistance should be as high as 8,000 to 10,000 ohms it is positive evidence that the nasal packing is inadequate.

The direct current is then turned on slowly to the desired strength. One hundred (100) milliampere-minutes, that is, 10 milliamperes for 10 minutes has been found to give good results. However, the strength of the current may be varied according to circumstances, and as dictated by experience.

The immediate discomfort to the subject is negligible and consists in having the nasal cavities tightly packed and a metallic taste as the current reaches its maximum. The after-results are more troublesome. Soon after the completion of the treatment there ensues a period varying, usually from eight to twelve hours, when there is an intense burning sensation in the nose, eyes and face accompanied by some pain and headache. In addition there is frequently a marked nervous reaction, the extent of which is dependent on the temperament of the subject.

The treatment has been developed primarily for the alleviation of hay fever and seems to be particularly applicable to those vasomotor disturbances of the nasal mucous membranes which are to-day usually classed as allergic.

Our experience, so far, has consisted in the treatment of sixteen subjects with a conservative general estimate of 85.6 per cent of relief.

Eight of these suffered from hay fever and in this group there was 85.6 per cent of relief. One subject suffering from fall hay fever was treated in June with the result that he was entirely free of symptoms for the first half of the season, after which the usual symptoms intervened

*Tobey, Harold G. — Surgeon, Massachusetts Eye and Ear Infirmary. For record and address of author see "This Week's Issue," page 240.

with their usual severity. All patients treated after the onset of the symptoms were free of all discomfort thereafter.

Eight were the victims of an hyperesthetic rhinitis and among these, three were also sufferers from asthma. Two of the latter had already been relieved of asthma before the treatment was instituted, while in the third there was no relief following the treatment.

In this group it will be noticed that while there was 83.7 per cent of immediate relief, the time element begins to make itself manifest.

Case 10 suffered from asthma, urticaria and hyperesthetic rhinitis for two years when first seen two years ago. She has been relieved of asthma now for one and a half years, and of the urticaria for one year. All efforts at relieving the rhinitis had proved of no avail up to May 1934 when the Warwick treatment gave complete relief for six months. The second treatment has just been completed so that the result is not yet available. The causative agent seems to be heavy perfumes which she has no way of avoiding since her livelihood depends upon the teaching of music.

Case 11 experienced about 50 per cent of relief for a period of two months.

Case 9 has had 50 per cent relief for the full six months following the treatment.

The other cases have experienced immediate relief but a sufficient time has not as yet elapsed for any final decision.

The change in the appearance of the mucous membrane is as striking as the relief from symptoms. Following the stage of primary congestion, the mucous membrane takes on the color of the normal healthy lining which is in great contrast to the pale, boggy appearance before treatment.

Case	Age	Dates of Ionization	For What Was Ionization Used? H. F.	H. R.	Asth.	Duration of Symptoms	Result of Ionization	Remarks
1	23	June '34	*			2 yrs.	50%	Fall hay fever treated in June
2	60	Sept. '34	*			40 yrs.	100%	
3	40	Aug. '34	*			Unknown	100%	Relief Total 16 85.6%
4	55	Aug. '34	*			Unknown	85%	Hay F. 8 87.5%
5	7	Oct. '34	*			5 yrs.	75%	Hyp. R. 8 83.7%
6	12	Oct. '34	*			3 yrs.	100%	
7	55	Sept. '34	*			Unknown	100%	
8	16	Aug. '34	*			10 yrs.	90%	
9	50	May '34		*		10 yrs.	50%	
10	48	May '34		*	*	4 yrs.	100%	Asthma, urticaria H. R. relieved 6 mos.
11	43	Aug. '34		*		3 yrs.	50%	Two months
12	45	Oct. '34		*	*	17 yrs.	100%	No relief to asthma
13	20	Aug. '34		*		3 yrs.	75%	
14	18	June '34		*		3 mos.	100%	
15	52	Aug. '34		*	*	10 yrs.	95%	Asthma, Migraine, Urticaria
16	40	Nov. '34		*		3 yrs.	100%	
			8	8	3			

CONCLUSIONS

1. The method is efficient for the control of hay fever if instituted after the onset of the symptoms. We feel, however, that desensitization should be the method of choice.
2. The immediate results in hyperesthetic or vasomotor rhinitis are excellent. The duration of these results is as yet unknown.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 21311

PRESENTATION OF CASE

A thirty-eight year old Chinese-American waiter entered complaining of abdominal pain.

During the past year the patient had gas pains localized to an area about four inches in diameter just below and to the right of the umbilicus. The pain was constant, occasionally sharp, and never radiated. At times it produced nausea but never actually caused him to double up or vomit. Three months before admission it became more severe and more constant. It bore no relation to meals but if he ate a hearty meal it would become more severe. His appetite, however, remained good. The pain was relieved in three ways: (1) pressure over the painful area, following which he could feel and hear the gurgle of gas passing; (2) a bowel movement; (3) lying on his right side with his knees slightly flexed. There had been no change in his bowel habits except that he had used mineral oil for the past three weeks on the advice of his physician. There was no pain associated with his bowel movements, nor had he ever noticed any bloody, tarry or clay-colored stools. During the past three months before admission he occasionally felt feverish and stated that on one afternoon a week before admission he had had a temperature of 102°. He had never had cough or hemoptysis. He had frequent night sweats during this illness. He had lost approximately thirty pounds in weight during the past three months. His present weight was about ninety-five pounds.

His father and mother, both over seventy, were living and well. One brother and sister were living and well.

He had been married twenty-one years. His wife and three children were living and well.

There was no history of carcinoma, diabetes or tuberculosis. Except for an appendectomy fourteen years before admission the past history is non-contributory.

Physical examination showed a thin man in no acute distress. There was evidence of marked loss of weight. The heart and lungs were negative. The abdomen was soft and very tympanitic except in the right lower quadrant, which was completely occupied by a firm, elastic, slightly tender, fixed, irregular mass. The right lower quadrant appendectomy scar was fixed to the mass. The right thigh could not be fully extended without pain. There was one small gland in the right axilla. His fingers were slightly clubbed. The blood pressure was 110/60.

The temperature was 99.2°, the pulse 95. The respirations were 25.

Examination of the urine was negative. The blood showed a red cell count of 4,600,000, with a hemoglobin of 85 per cent. The white cell count was 13,000, 77 per cent polymorphonuclears. A Hinton test was negative. A barium enema passed without delay to a point in the transverse colon just distal to the hepatic flexure. At this point a loop of small bowel was observed to fill. There was a definite obstruction at this point to the passage of barium onward into the colon but the loops of small bowel continued to fill. A small amount of barium did pass on to outline poorly a very spastic cecum and ascending colon. The mass was found to overlie the constricted area, which was three inches in length. The edges of this constriction were shelved and the margins showed marked irregularity. A chest film was negative. A flat abdominal film was negative.

On the third day through an incision just inside the right anterior superior spine an abscess cavity was broken into by blunt dissection without entering the peritoneal cavity. A tube was sutured into the cavity and the wound closed. A culture of this abscess showed bacillus coli and non-hemolytic streptococcus. He ran a very slightly septic temperature, ranging between 99° and 101°. Approximately three weeks after the first operation the incision in the flank was enlarged posteriorly in order to give him adequate and more dependent drainage. Ten days later laparotomy was performed.

DIFFERENTIAL DIAGNOSIS

DR. LELAND S. MCKITTRICK: We might stop for the moment and review the information we have been given down to the laboratory examination.

"During the past year the patient had gas pains localized to an area about four inches in diameter just below and to the right of the umbilicus." Three months before admission the pain became more severe and continuous. I do not know what the writer means by "gas pains". According to my own conception it is a pain that comes and goes, more like colicky pain. The more you talk about steady pain,—I dissociate it a little bit from the so-called gas pains—the more we think of an inflammatory process. It is conceivable and quite likely that both are associated and that he may have something interfering with the normal peristaltic activity, presumably of the large bowel, because the level of the pain is below the level of the umbilicus and in addition he may have a localized inflam-

matory process somewhere in the right abdomen giving the more constant pain and soreness.

His procedures for getting relief are interesting and significant: "(1) Pressure over the painful area following which he could feel and hear the gurgle of gas passing." I do not know just how much importance to stress on the pressure of the hand over that area. I presume his relief of pain came in some way during manipulation, causing him to pass gas. "(2) A bowel movement." That is a perfectly definite, concrete thing. He has pain, has a bowel movement, and then has relief from pain. That to me means that he probably has an obstructing lesion in the large bowel. "(3) Lying on his right side with his knees slightly flexed." The only significance that I can put on that is that he probably has an inflammatory process. When he lies down and relaxes the abdominal muscles by flexing his knees, he gets some relief from the discomfort he would otherwise have from the more tense muscles. To me that suggests that in addition to the obstructing lesion of the bowel there must be some inflammatory process present. There is very little in the rest of the present illness except the fact that he has had fever. He knows he had a fever of 102° on one occasion and he has felt feverish at other times. This is wholly in keeping with the conception that there is present an inflammatory process. His loss of weight fits in with either or both.

The next important thing is in the physical examination. The entire right lower quadrant is completely occupied by a firm, elastic, slightly tender, fixed, irregular mass. He has an appendectomy scar which seems to be fixed to the mass; also the right thigh cannot be extended without pain. Whether that means that stretching his ileopsoas is the cause of the pain or simply means that in flexing the thigh there is a relaxation of all the abdominal muscles and relief of pain for that reason, I do not know. But I think it is suggestive again of the presence of an inflammatory process. I do not believe that one can thus far draw any conclusion other than to say that it would seem as if this man had an inflammatory process involving the large bowel in such a way as to give him some mechanical interference with a normal bowel activity.

The blood examination gives us no help. Frequently in a malignant disease of the right colon a marked secondary anemia is present. On the other hand one can have it without the secondary anemia so that that picture does not give me any great help, although I confess I am surprised that a man who has been sick as long as he has, who has lost thirty pounds and has been running a fever, should have as high a red cell count and hemoglobin.

He has by barium enema a definite obstruction at the proximal portion of the transverse colon; when the barium gets around to that point the small bowel seems to fill, the mass overlies the constriction, and the lumen of the bowel presents a shelving. That must mean an elevated portion within that area where it is constricted. The constriction is about three inches in length. The negative chest film may or may not be of interest in relation to a subsequent discussion of tuberculosis as a possibility. I mention it now because I think it can be dismissed. Tuberculosis of the bowel may occur in the absence of x-ray evidence of disease of the lungs just as well as in its presence. Diffuse enteritis is practically always associated with tuberculosis of the chest but localized tuberculosis that we see in the large bowel is not.

As we come down to the laboratory notes, I picture a process in the right colon which is a combination of an obstructive with an inflammatory process. That is, he may possibly have had some type of inflammatory process which had constricted and obstructed the bowel or he may have had some type of lesion within the bowel which had perforated subacutely and had given him an inflammatory process secondary to the lesion of the bowel itself. A barium enema gives us very definite information. It localizes a lesion just distal to the hepatic flexure, that is the proximal portion of the transverse colon. I presume this man was small and thin, was very light probably, and had a very low transverse colon so that the mass could swing around and seem to be lower down in the bowel than it actually was. The other interesting thing in the x-ray is that as the barium reaches the obstruction the small bowel fills. There is only one interpretation that I can intelligently put on that. Whatever the process is, there must be an opening between the transverse colon at that point and a loop of small bowel which must be adherent to the lesion. In other words, we have an obstructive process involving the proximal portion of the transverse colon to which a loop of small bowel is adherent and between these loops a spontaneous anastomosis has taken place.

What are the conditions that one has to consider in attempting to come to some conclusion as to what the process is? Of course the thing that immediately comes up is carcinoma. I cannot from anything that is in the story exclude a carcinoma of the transverse colon with involvement of a loop of small bowel with secondary abscess. That is a possible explanation for the process. There is nothing about it which is not easily and entirely in keeping with such a diagnosis. Tuberculosis is the next, I think, and most likely. I think it is quite possible that the formation of a fistulous opening between this process and the adjacent coil of small bowel may be more in keeping with a tuberculous lesion than with malignant disease. As

we have already said, a negative chest x-ray means nothing. He has had a suggestion of fever with this for some little time, which might suggest strongly that it is of tuberculous origin rather than malignant disease. According to the description of the x-ray here—and I am quite willing to admit I do not know too much about tuberculosis of the large bowel—but my conception of tuberculosis of the large bowel is not quite in keeping with a constriction the edges of which are shelved. The shelving of the edges of the constriction to me more definitely suggests the possibility of a new growth in the bowel than it does the more diffuse process that one gets with tuberculosis. On the other hand I cannot completely and intelligently exclude it. There are certain other things that one thinks of. I have seen two cases of actinomycosis of the right side following appendectomy, but in each of the cases the interval between appendectomy and actinomycosis was a matter of a few weeks or months. There is an interval of fourteen years here so that the appendix cannot be a source for it. The description of an elastic mass is wholly out of keeping with the board-like mass one gets in actinomycosis and I think that can be dismissed merely after the mentioning of it.

Lymphosarcoma that one sees in the large bowel again must be mentioned and certainly is a possibility. I do not think that that is what it is. Most lymphosarcomas we have seen have not gone on to abscess formation in this way, and while again it is a possibility we may exclude it. Some time ago I happened to discuss at one of these conferences a large intestine case and about two months afterwards I had a very nice letter from a doctor in Nigeria who described a patient with a mass in relation to the right colon due to entameba histolytica. This patient is a Chinaman but I believe he has been in this country all of his life. He has not diarrhea, although this does not exclude it.

The other condition that one has to consider is the localized inflammatory process usually termed localized or regional ileitis. This lesion may involve the large bowel. It shows a great tendency to become adherent to adjacent structures, a fistula frequently forms and it is perfectly conceivable that such a process may be present in this man. It is a very unusual condition and, although there is no diagnostic x-ray picture and while none of us have seen many of these conditions, particularly in the large bowel, I do not think one would expect the constriction to have shelved margins. Therefore I would not be content to make that diagnosis.

It seems to me that the reasonable conclusion is that this man has either a carcinoma or tuberculosis of the beginning of the transverse colon with a loop of small bowel adherent to it and with a communication between, and with the large mass surrounding the entire area largely inflammatory in character.

Clinical Discussion

Dr. Arthur W. Allen: This man was very ill when he came in and had obviously been ill for a long while. He had this enormous mass in his right side which was a good deal more tender I think than the history would lead you to believe. An enthusiastic service had a barium enema done before we saw the patient. We feel that barium enemas perhaps may be dangerous in large bowel lesions in the acute stage with obstruction and although it did this patient no harm and contributed some very valuable information I think perhaps we ought to mention the fact that it is not a good policy to do a barium enema routinely until at least you have had a flat plate of the abdomen. We felt that whatever the original process was in the colon that there was no question he had an abscess in the region of the cecum. So, in spite of any suspicion that we might have as to the original lesion, an incision and drainage of the abscess was done. He responded only moderately well to this operation. His temperature did come down at first but in a few days started to rise again. We were getting quite discouraged about him and finally decided that the only thing to do was redrain his abscess at a lower level. This gave a very good result evident in his chart and general condition, so that ten days after the second drainage it was possible to go in through a clean incision and resect the entire lesion. As you might imagine it was very adherent to surrounding structures, involving the cecum, ascending colon, terminal ileum, and extending into the psoas muscle, parietal peritoneum, etc. It was possible to remove it all and do a lateral anastomosis and the patient was finally discharged some twenty days after the last operation to a convalescent hospital.

Preoperative Diagnoses

First Operation: Pericecal abscess. Carcinoma of hepatic flexure.
Second Operation: Pericecal abscess.
Third Operation: Carcinoma of the cecum and transverse colon.

Dr. Leland S. McKittrick's Diagnoses

(1) Carcinoma or (2) tuberculosis of the transverse colon with involvement of the ileum.

Pathologic Diagnoses

Tuberculosis of colon and ileum.
Fecal fistula.

Pathologic Discussion

Dr. Benjamin Castleman: The surgical specimen consisted of the entire ascending colon

and a portion of transverse colon and terminal ileum. The anastomosis described by the roentgenologist was a surgical one and not spontaneous as one might infer from the lack of any history. Apparently at the time of his appendectomy fourteen years before, the surgeon resected a portion of cecum and anastomosed the terminal ileum to the ascending colon. What Dr. Allen did was to repeat this first operation higher up, i.e., resect the blind end of the ascending colon including the ileocolic anastomosis and perform another ileocolic anastomosis.

Almost the entire section of colon is markedly thickened and constricted. The mucosal surface shows an almost annular zone of irregular ulceration which corresponds to the shelving described by x-ray. This is especially marked in the distal portion. There is also ulceration of the ileum at the point of anastomosis and at the base of this region there is a fecal fistula that apparently had drained through the psoas muscle and from which a culture of bacillus coli was obtained. Histologic examination showed tuberculosis. This case apparently belongs to the hypertrophic type of intestinal tuberculosis which often shows ulceration with surrounding areas of elevation. It is not the acute form of ulcerating enteritis that is usually associated with pulmonary infection.

DR. ALLEN: We thought that was a spontaneous anastomosis and the x-ray people did commit themselves with a diagnosis of carcinoma as the first choice and tuberculosis as the second. I thought it was carcinoma.

DR. RALPH ADAMS: Dr. Langdon Parsons and I went over fifty-two cases of tuberculosis of the cecum from 1924 to 1933. Of these, twenty-four were proved pathologically. We were directed to this study by a case that Dr. Parsons had in the Baker Memorial in which it was difficult to differentiate carcinoma of the cecum and tuberculosis. In fifty-two cases of carcinoma of the cecum during the same period four had a preoperative diagnosis of tuberculosis. The most common symptom was pain referred to the region of the umbilicus. An irregular mass with tenderness was found in half of the cases and most of them were fixed to the right lateral wall. A minor point in distinction was that the red blood cell count was not below 3,800,000 in any of the cases of tuberculosis as contrasted with the carcinoma cases, in which the counts were almost all below 3,500,000. A positive guaiac on the stools appeared only five times. This we thought was due to the fact that most of our cases fell within the older groups which tend to have constriction and absence of blood, as against diarrhea and bloody stools in the younger groups. The x-ray findings consistently reported spasm of the cecum. These findings were spoken of as a standard defect both in the hospital reports and in the literature. The attempt to confirm the barium enema by a gastrointestinal series almost consistently failed and in several cases the diagnosis was not made at the first entry. The most common error was the interpretation of the films as carcinoma of the cecum. We found that one-third of the patients in the age group over thirty-five who had the disease had it in the absence of clinical or x-ray evidence of tuberculosis in the chest and only one-third of the cases had clinical symptoms referable to the lungs or chest.

CASE 21312

PRESENTATION OF CASE

First Admission. A thirty-five year old Italian laborer entered complaining of right lower quadrant pain.

Approximately one year before admission he noticed that he was bleeding from his rectum. This hemorrhage continued for four or five days and then disappeared for a period of a month. There was no pain and no other symptoms except for moderate constipation. The hemorrhage continued to appear at intervals until three months before admission, at which time it became continuous, more marked at night. Associated with this increased melena there was severe tenesmus, often as much as thirty times a day, each attempt being rewarded by a slight show of watery fecal material and some blood. During the past three months there had been intermittent pain, often very severe, which was localized to the right lower quadrant but at times was generalized throughout the abdomen and was often relieved by a bowel movement. There was some pain upon urination, especially on the right side of the scrotum. During the past year he had lost about fifteen pounds in weight.

His family, marital and past histories are non-contributory. There was no history of carcinoma or tuberculosis.

Physical examination showed a thin, undernourished man with evidence of recent loss of weight. His teeth were dirty and carious; there was moderate pyorrhea. The chest showed a few prolonged inspiratory and expiratory, sibilant and sonorous râles. There was no dullness. The breath sounds were not prolonged. On rectal examination there was a firm, irregular, fixed annular mass well within the reach of the finger. The finger, however, could be passed through the lumen but the top of the growth could not be reached. The blood pressure was 106/64.

The temperature was 99.6°, the pulse 65. The respirations were 20.

On the day following admission abdominal exploration revealed a firm, annular mass in the upper portion of the rectum. The liver was free from metastasis. Upon incision the perineal reflection from the rectum was found to have extensive glandular enlargements. It was thought unwise to attempt resection of the

growth. The sigmoid was then delivered through the wound and a colostomy performed. Histologic examination of a lymph node and a fragment from the rectum showed no evidence of malignancy.

He had a stormy postoperative convalescence and was finally discharged two weeks after operation to the Huntington Hospital for radium treatment.

Second Admission, four years later.

Following discharge he reported to the Huntington Hospital every two or three weeks for radium treatment over a period of six months. A histologic examination of a piece of tissue from the region of the tumor removed at the Huntington Hospital showed no evidence of neoplasm. He did well for the remainder of this interval until a few days before this admission, when he complained of slight pain in the right lower quadrant. There had been no nausea or vomiting but he had bled occasionally from his colostomy. His stools, however, were normal in consistency.

Physical examination showed a hernia in the lower end of the abdominal wound. The abdomen was soft. In the right lower quadrant there was a slightly tender movable mass approximately 4 centimeters in diameter.

His chart was normal.

Examination of the urine was negative. The blood showed a white cell count of 13,800.

On the third day through a right oblique abdominal incision a dilated, thickened adherent appendix was removed. Exploration revealed that the mass felt before operation was in the cecal wall and mesentery forming a tumor approximately 5 by 3 centimeters. The rectal tumor had completely disappeared except for induration in the pelvic floor. At the point where the rectum passed through this there was a narrow stricture. The liver was negative. The ileocecal glands were enlarged and one was removed. Histologic examination of the glands and appendix showed lymphoid hyperplasia and healed appendicitis respectively.

He did fairly well postoperatively and was discharged two weeks later.

Final Admission, fifteen months later.

Following the last admission the mass in the right lower quadrant enlarged and receded in size and because of this the patient thought it was gas. The pain, however, continued as a dull steady ache. One week before admission he began having colicky pains in the right lower quadrant which lasted about fifteen to twenty seconds and recurred every five minutes. His colostomy had continued to function satisfactorily although he did have some constipation close to the colostomy opening. Abdominal examination showed, in addition to the colostomy and right lower quadrant scar, a hard, irregular, movable, egg-sized mass apparently intraäbdominal but located just beneath the appendectomy scar.

The temperature was 97°, the pulse 80. The respirations were 20.

Examination of the urine was negative except for an occasional white blood cell and many brown granular casts. The blood showed a red cell count of 6,000,000, with a hemoglobin of 80 per cent. The white cell count was 10,900, 79 per cent polymorphonuclears. Two stool examinations gave negative guaiac tests. A smear was negative for tubercle bacilli. A Hinton test was negative.

X-ray examination was unsatisfactory because the patient expelled the enema.

On the fourth day operation was performed, and the cecum, ascending colon and terminal foot of ileum were resected. He did poorly postoperatively, developed signs of bronchopneumonia and died on the fifth postoperative day.

Differential Diagnosis

Dr. Arthur W. Allen: This is such a typical story of carcinoma of the rectum that, except at these clinico-pathologic conferences, almost any other possibility would be precluded.

Of course this is a fairly young man, only thirty-five, but carcinoma of the rectum may happen in the twenties and I believe cases have been reported in the teens; thirty-five is not too young an age to have such a disease appear. He had been well until he started to bleed from his rectum. He bled a little for a few days and then stopped and then bled intermittently up to three months before he came in, when he began to bleed daily and had tenesmus requiring relief at the toilet many times a day. That is a very typical story for carcinoma of the rectum. This growth was easily felt by the examining finger and some lumen was still present, but it was not possible to get a proctoscope or finger through it. Such tumors are hard and indurated and frequently there is so much infiltration, either inflammatory tissue or malignant disease, around them that it is often impossible to reach the top of the growth. The pain on urination would lead one to suspect that the metastases had perhaps encroached upon the urinary tract, and further confirms the suspicion of a very advanced carcinoma of the rectum with marked pelvic metastases.

He had lost fifteen pounds in weight, probably due to diarrhea.

At operation the liver was free of metastasis. It is not at all uncommon in carcinoma of the rectum where the pelvis may be full of metastatic nodules to find that the disease has not reached the liver. Obviously the operator felt that the tumor was not removable and did a colostomy above the growth which in itself probably would not have affected the tenesmus very much. A point which I think one might speak of with colostomy above a low growth, one that is creating constant desire to defecate, is that colostomy does not always relieve tenesmus very

much. It does help if in addition to the colostomy one divides the sphincter muscles so that there is a continuous discharge, thus eliminating the urge to evacuate the rectum.

The fact that we had a negative pathologic report from the lymph node and from a biopsy from the rectum seems a little queer and makes us wonder; that is, if it was negative so far as malignant disease is concerned, and makes us wonder what type of lesion we could be dealing with if it is not carcinoma. I am not worried about the biopsy from the rectum because in these growths where there is more or less stricture it sometimes is not possible to get a specimen from the growth itself. One may easily take a bit of mucous membrane where it is folded over the edge of the growth and that naturally would not show any of the disease, for this reason occasionally rectal biopsies do turn out to be negative so far as malignant disease is concerned even if the diagnosis is clinically evident. The fact that the lymph node was not involved makes me a little bit more suspicious because one would expect the lymph node to show metastatic disease. However, I believe that I will so far have to conclude that this man had carcinoma of the rectum. He was treated at the Huntington Hospital with radium. I believe that he would not have had six months of radium treatment at the Huntington Hospital if the Huntington Hospital as well as this hospital had not thought that this man had malignant disease.

"A histologic examination of a piece of tissue from the region of the tumor . . . showed no evidence of neoplasm." One wonders how that specimen was removed, whether from the tumor itself or whether again they got a piece of normal mucosa.

The dilated appendix is an important factor and means that he had obstruction beyond the cecum somewhere because the appendix is distended with gas only where there is obstruction beyond the cecum.

It is a little disturbing that we have no note about the rest of the abdominal exploration. To what was his large bowel pain due? Was it due to a poorly functioning colostomy which might have given him backing up in the rest of the colon or has he a lesion which was not palpated somewhere else in his colon between the cecum and colostomy. We know that he had bled several times through the colostomy. If that is true it is an important statement but practically all colostomies have a tendency to ooze a little bit from the exposed mucosa at times. Often there will be a little blood on the dressing. It may be an accurate story. If it is accurate, it is important, because if he has bled he has a lesion somewhere else in his colon. The histologic examination of the colon showing lymphoid hyperplasia is again not necessarily against the fact that this lesion in the bowel is carcinoma. Obviously the operator felt that this was not a malignant lesion in the cecum and felt it was an inflammatory lesion or he would have prescribed something more radical than appendectomy for relief of the tumor of the cecum in the presence of what appeared to be an apparently healed lesion in the pelvis.

"In the right lower quadrant there was a slightly tender movable mass approximately four centimeters in diameter." One would draw the conclusion from this description that this mass in the right lower quadrant had not increased tremendously in size during the past fifteen months, which is somewhat surprising.

His red blood cell count is put down in large figures, 6,000,000, and the hemoglobin 80 per cent, which coincides perfectly well with Dr. Adams' report on tuberculosis of the cecum but not very well with carcinoma of the cecum. We expect anemia associated with carcinoma of the cecum.

We have, then, a patient with a perfectly typical story for carcinoma of the rectum who apparently, after a colostomy, was successfully treated with radium so that he had a respite of four years. I am not sure how many inoperable carcinomas of the rectum are successfully treated with radium but my impression is that they are not very common. The percentage I am sure is very much less than the percentage of cures of carcinoma of the cervix treated by radium. Then he comes back four years afterwards apparently with a mass in the region of the cecum which again is negative to biopsy so far as malignancy is concerned and if we are to believe his story he probably has bled some from the colostomy. The statement that this growth in the region of the cecum did not seem to increase greatly in fifteen months may not give us a true conception of affairs because it may have been a slowly growing lesion of one sort or another. I would feel that this man had probably multiple polyps in his large bowel. The first one to become malignant being in the rectum, afterwards another lesion in the colon, probably independent of the original tumor. Multiple malignant lesions in the colon are not too uncommon developing on polypi, so I should feel that that would perhaps explain the story better than any other condition and that he had in addition to this mass in his cecum something in the colon between that and his colostomy. I do not think that tuberculosis could possibly explain this picture. Time is short and I cannot go into all the details on that. I do not believe lues would explain it. I cannot imagine any type of inflammatory condition that would explain the original condition. It is possible that the second lesion could be non-specific granuloma (regional ileitis) but I think that is unlikely. Here is a man with a carcinoma tend-

ency possibly, and there is no reason why he should not have multiple lesions. Lymphosarcoma has to be mentioned as a possibility and that might be the second best bet, but I should adhere to the diagnosis of carcinoma as the first.

Clinical Discussion

Dr. Leland S. McKittrick: I did not remember having seen this man until I was just shown the record. I saw him in 1924 and treated him with radium and I remember him because I thought he had a carcinoma of the rectum and later all evidence of disease disappeared, but he could not be recorded as a cure because we did not have a positive pathologic report. He had a fixed growth, very hard, which I had never at that time seen cured by radium, nor have I since.

Clinical Diagnosis

Preoperative: Hypertrophic tuberculosis of the cecum.

Dr. Arthur W. Allen's Diagnosis

Carcinoma of the large bowel (multiple).

Anatomic Diagnoses

Amebic granuloma of the cecum.
Operation wounds: Resection of the cecum.
Ileocolic anastomosis.
Colostomy.
Bronchopneumonia.

Pathologic Discussion

Dr. Benjamin Castleman: The preoperative diagnosis at his last operation was hypertrophic tuberculosis of the cecum. The surgeon found a firm tumor which felt like carcinoma involving the cecum. The tumor was firmly adherent in the right flank. No metastases to the liver or tubercles on the peritoneum were seen. Some of the mesenteric glands were enlarged. The surgeon's postoperative diagnosis was also tuberculosis.

The specimen consisted of the cecum, ascending colon and a portion of terminal ileum. Almost the entire cecum was replaced by an annular, very firm, ulcerated granulomatous mass about seven centimeters in length. The cut surface of the tumor was hard, white and fibrous. Histologic sections of the wall of the ulcerated area showed a layer of granulation tissue, the superficial portion of which was necrotic, while the deeper portions and the dense fibrous tissue below showed a marked chronic inflammatory reaction, the predominant cell being the eosinophil, although there were moderate numbers of lymphocytes and plasma cells. In a few places along the inner margin of the necrotic granulation tissue definite entamebae histolyticae were seen, one containing many red blood cells.

This case is quite similar to the condition called "amebic granuloma" by Gunn and Howard* of Stanford University. They reported three cases of amebic granulomatous lesions of the large bowel, all giving symptoms, signs and x-ray appearances of carcinoma, and all called carcinoma clinically. Two of their cases showed involvement of the cecum and the other of the transverse colon. In this part of the country where amebic infections are so rare one almost always thinks only of the dysenteric form and rarely hunts for amebae in the stools except in cases of severe diarrhea. It certainly was not suspected in this case and no search of the stools was ever made.

This case also brings up the question of the reported cures of inoperable cancer of the colon by x-ray and radium. I feel certain that some of them, especially those in which a biopsy was not done, might well have been amebic or other types of granuloma.

Unfortunately we were not able to examine the rectum, but there is little doubt that the original lesion there was identical.

*Gunn, H., and Howard, N. J.: Amebic granulomas of the large bowel. J. A. M. A. 97:166, 1931.

The New England
Journal of Medicine

SUCCESSOR TO
THE BOSTON MEDICAL AND SURGICAL JOURNAL

Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

COMMITTEE ON PUBLICATIONS

R. I. LEE, M.D., *Chairman* HOMER GAGE, M.D.
R. B. OSGOOD, M.D. R. M. SMITH, M.D.
F. H. LAHEY, M.D.

EDITORIAL STAFF

REID HUNT, M.D. STEPHEN RUSHMORE, M.D.
JOHN P. SUTHERLAND, M.D. HANS ZINSSER, M.D.
GEORGE R. MINOT, M.D. BENJAMIN WHITE, PH.D.
FRANK H. LAHEY, M.D. HENRY R. VIETS, M.D.
SHIELDS WARREN, M.D. ROBERT N. NYE, M.D.
GEORGE L. TOBEY, JR., M.D. ROBERT M. GREEN, M.D.
C. GUY LANE, M.D. CHARLES C. LUND, M.D.
WILLIAM A. ROGERS, M.D.

WALTER P. BOWERS, M.D., *Managing Editor*

ASSOCIATE EDITORS

GEORGE G. SMITH, M.D. WILLIAM B. BREED, M.D.
JOSEPH GARLAND, M.D.

THE NEW HAMPSHIRE MEDICAL SOCIETY
PUBLICATION COMMITTEE

CARLETON R. METCALF, M.D. HENRY H. AMSDEN, M.D.
WARREN H. BUTTERFIELD, M.D.

THE VERMONT STATE MEDICAL SOCIETY
PUBLICATION COMMITTEE

WILLIAM G. RICKER, M.D. C. F. DALTON, M.D.
L. H. ROSS, M.D.

SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $8.52 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 8 Fenway.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The New England Journal of Medicine, 8 Fenway, Boston, Mass.

THE PREVENTION OF POSTVACCINATION ENCEPHALITIS

POSTVACCINATION encephalitis has been a subject of considerable interest in recent years, and occasional cases have been reported in the American literature; seventy-five in all, according to Bauer[1], including his own case, a six-year old girl who made a complete recovery. The course of the disease is stormy and the prognosis grave, although, when not fatal, complete mental and physical recovery is the rule. This seems surprising in view of the pathologic picture, consisting of adventitial and periadventitial round cell infiltrations distributed throughout the brain and cord with myelin degeneration about the smaller vessels.

Of especial interest, however, is Bauer's advice as to the prevention of postvaccination encephalitis, divided, according to clinical evidence, under three heads. First, the rarity of postvaccination encephalitis in infants under one year of age indicates that infants are not apt to be sensitive to virus in these early months of life, and the same holds true of secondary vaccinations at any age.

Secondly, if further desensitization to virus has been accomplished by other immunizing agents, such as diphtheria toxoid, there is still less chance of developing encephalitis. Vaccination performed too soon after the toxoid infection, however, particularly if alum precipitated toxoid is used, will probably not be successful; at least a month must be allowed to elapse.

Thirdly, the single short scratch or puncture method of vaccinating should be employed, as this allows the minimal effective amount of virus to enter the skin and desensitize the individual before its multiplication reaches the amount of the large doses administered by other methods.

REFERENCE

1. Bauer, E. L.: J. Pediat. 6:512 (April) 1935.

THE BOSTON CITY HOSPITAL

THE way in which a large and loosely organized public institution may be exploited for private gain is suggested by the recent charges of the Boston Finance Commission against the City Hospital. Apparently there have been gross waste and theft of materials, equipment and food on a fairly large scale, and charges of lax discipline, excessive personnel and political interference in the hiring of employes have been made, whether or not those charges can be substantiated.

Certain figures, however, are difficult to explain. Thus, it is stated that Bellevue Hospital in New York, feeding 1253 more persons daily than the Boston City Hospital, annually uses 50,000 pounds less meat and 25,000 pounds less butter. The Boston hospital has 13 employes for every 10 patients, as compared with 10 employes to 13 patients in similar institutions in other cities.

Further findings of the commission are as follows:

In order to stop the present thieving and to prevent interlopers from having the run of the hospital and use of the employes' dining rooms, a stricter control of the entrances and exits should be established.

Automobile parking on the grounds constitutes a fire menace.

Excessive personnel is due to the fact that many temporary employes furnished from the mayor's office are unfit or refuse to do their work, necessitating additional help. (This statement the mayor has denied.)

The labor turnover is greater than in any other city department except, perhaps, the department of public works.

Long and unwarranted delay in the opening

of the new $400,000 kitchen unit was partly responsible for the waste in foodstuffs.

A business manager ought to be appointed with full responsibility and control over the business administration of the hospital.

Authority over and responsibility for the employment of employes other than the surgical and medical staff should be vested in the various heads of the division of administration under the general supervision of the business manager.

Efforts should be made to bring about a greater coördination of the operation of the various departments of administration, and also such new construction as may from time to time be in process.

A system of adequate and accurate records for the business administration of the hospital should be established and maintained.

The findings of the commission deserve serious consideration, and it is probable that they are substantially correct. There is no question that the trustees of the hospital are a body of able and honest men, receiving inadequate recognition and functioning to the best of their ability with inadequate authority to prevent abuses or effect a reorganization.

The only real solution of the City Hospital's problems is the appointment of an able administrator who will be free from political influence and vested with enough power to keep the institution above politics.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

ELEY, R. CANNON. M.D. Department of Medicine, University of Virginia, 1925. Associate in Pediatrics and Communicable Diseases, Harvard Medical School and Harvard School of Public Health. Associate Visiting Physician, Children's Hospital. His subject is "The Control of Measles." Page 195. Address: 319 Longwood Avenue, Boston, Massachusetts.

MCDONALD, FRANCIS C. A.B., M.D. Harvard University Medical School 1929. Assistant to the Physician-in-Chief, Boston Floating Hospital. Instructor in Pediatrics, Tufts College Medical School. Assistant Physician, Children's Clinic, The Boston Dispensary. His subject is "Whooping Cough." Page 198. Address: 370 Longwood Avenue, Boston, Massachusetts.

ANDERSON, GAYLORD W. A.B., M.D. Harvard University Medical School 1928. Deputy Commissioner and Director of the Division of Communicable Diseases, Massachusetts Department of Public Health. Assistant in Public Health Administration, Harvard School of Public Health. His subject is "The Present Status of Scarlet Fever Prevention." Page 203. Address: Room 546, State House, Boston, Massachusetts.

ROBINSON, ELLIOTT S. B.A., Ph.D., M.D. Yale University School of Medicine 1918. Director, Division of Biologic Laboratories, Massachusetts Department of Public Health. His subject is "The Control of Diphtheria." Page 208. Address: 375 South Street, Jamaica Plain, Massachusetts.

SMITH, RICHARD M. M.D. Harvard University Medical School 1907. Sc.D. Assistant Professor of Pediatrics and Child Hygiene, Harvard Medical School and School of Public Health. Visiting Physician, Infants' Hospital, Boston. Associate Visiting Physician, Children's Hospital, Boston. He presents a "Summary" of the Symposium on the Control of Communicable Diseases. Page 211. Address: 66 Commonwealth Avenue, Boston, Massachusetts.

BARNES, J. ARTHUR. A.B., M.D. Harvard University Medical School 1899. F.A.C.S. Attending Surgeon, St. Vincent Hospital, Worcester. Address: 390 Main Street, Worcester, Massachusetts. Associated with him is

RICHMOND, EUGENE L. A.B., M.D. Cornell University Medical College 1929. Assistant Attending Surgeon, St. Vincent Hospital, Worcester. Address: 390 Main Street, Worcester, Massachusetts. Their subject is "Hyperinsulinism." Page 225.

NICKERSON, D. A. M.D. Tufts College Medical School 1933. Assistant in Pathology, Mallory Institute of Pathology, Boston City Hospital. His subject is "Brain Abscess as a Complication of Septic Pleuropulmonary Disease." Page 228. Address: Mallory Institute of Pathology, Boston City Hospital, Boston, Massachusetts.

TOBEY, HAROLD G. A.B., M.D. Harvard University Medical School 1911. F.A.C.S. Surgeon, Massachusetts Eye and Ear Infirmary. Visiting Otolaryngologist, Children's Hospital. Otolaryngologist, New England Baptist Hospital. His subject is "Experiences in Ionization of the Nasal Mucous Membrane." Page 230. Address: 270 Commonwealth Avenue, Boston, Massachusetts.

MISCELLANY

DECISIONS OF THE SUPREME JUDICIAL COURT

Appeal, by the insurer in proceedings under the workmen's compensation act, from a decree entered by order of Whiting, J., in the Superior Court.

Pierce, J. This is an appeal by the insurer from a decree entered in the Superior Court after certification from the Industrial Accident Board, by which payment for services of a physician, George

W. Blood, was ordered G. L. (Ter. Ed.) c. 152, s. 30 reads: "During the first two weeks after the injury, and if the employee is not immediately incapacitated thereby from earning full wages, then from the time of such incapacity, and in unusual cases, or cases requiring specialized or surgical treatment, in the discretion of the department, for a longer period, the insurer shall furnish adequate and reasonable medical and hospital services, and medicines if needed, together with the expenses necessarily incidental to such services. The employee may select a physician other than the one provided by the insurer; and in case he shall be treated by a physician of his own selection, or where, in case of emergency or for other justifiable cause, a physician other than the one provided by the insurer is called in to treat the injured employee, the reasonable cost of his services shall be paid by the insurer, subject to the approval of the department. Such approval shall be granted only if the department finds that the employee was so treated by such physician or that there was such emergency or justifiable cause, and in all cases that the services were adequate and reasonable and the charges reasonable."

The reported evidence which, in the main, is not disputed by the appellant insurance company, discloses the following facts: One Katherine Zombric on October 7, 1930, was twenty-two years of age and was then employed as an operative in the mill of the United Rayon Company of Fall River. On that day, shortly after she began work in the morning, her hair was "caught in a revolving shaft, and her scalp and back of neck were torn off." She was immediately taken to St. Anne's Hospital. This hospital was incorporated under the provisions of R. L. c. 125 "for the purpose of . . . maintaining . . . homes or hospitals for the care of persons who are ill, disabled, invalid or convalescent, and furnishing them with medical and surgical treatment, support and nursing, and of maintaining schools for the training of nurses." Shortly after the arrival of the employee at the hospital some one called Dr. George W. Blood "to go down to the hospital on an emergency as quickly as possible." Dr. Blood was a member of the staff of the hospital but was not "on service" that week. However, he was subject to call from the hospital for the treatment of emergency cases.

Dr. Blood testified that it was one of the conditions under which he took service at the hospital that he would be subject to calls by the hospital in the event of an emergency arising; that the case of the employee was an emergency case and that he was called by the hospital and not by the employee nor by any member of her family; that after he treated the employee at the hospital her father came to see him at noon time and he had some conversation with him; that he told the father all about the case, how long she would be in the hospital, and that he was perfectly free to get any doctor he wished; that the father's first reaction was that he wanted a specialist; *that Dr. Blood said that he specialized in surgery and the father told him to keep on and do the best he could with the case.* The evidence is not disputed by the insurer that the skin was torn over the right side and back of the neck, nor that the severity of the injury necessitated several skin graftings and manipulations of the neck muscles at different intervals nor that the entire recovery of the patient required several months' treatment. It is not disputed that the insurer has paid the compensation due the employee under the statute, as well as the bills for her hospital treatment from October 7, 1930, until November 7, 1931, when she was discharged from the hospital.

On October 14, 1930, Dr. Blood wrote the insurer, Arrow Mutual Liability Insurance Company, informing that company of the fact that Katherine Zombric had been injured at the United Rayon Mills on October 7, 1930. This letter stated the cause and the result of the injury and concluded: "This is a very serious and unusual accident and will probably require several months of treatment and possibly several skin grafting operations. I would appreciate a note from you authorizing such treatment." The insurance company on October 20, 1930, acknowledged receipt of Dr. Blood's letter in these words: "*The accident to the above employee was an extremely unfortunate affair, and we are eager to do everything to make possible her recovery. We know that Miss Zombric had been sent to the surgical service of St. Anne's Hospital, but had no knowledge as to what member of the hospital staff was in charge of this case. We are pleased to learn that the girl is being treated on your service, and we will be happy to cooperate with you in any way.* Several days after the accident the Mother Superior of the hospital called on Mr. Joseph A. Parks at the State House; and at that time we assured Mr. Parks that we would accept liability because of the hospital care. Dr. J. Newton Shirley of this office is usually in Fall River each week, and I am asking Dr. Shirley to keep in touch with you and to give you any assistance you may need." The record does not disclose that the insurer furnished any medical attention to the employee following its letter of October 20, 1930, nor that any physician other than Dr. Blood attended the employee. There is no direct evidence that the employee herself selected Dr. Blood as her physician nor that she authorized any other person to do so on her behalf, but the employee's acceptance of the medical services of Dr. Blood for many continuous months clearly warranted the inference, if it did not require a finding, that the employee ratified and adopted the action of her father in this regard.

It is not contended or found that there was in fact any contract between the insurer and Dr. Blood to pay for the services rendered. The contention of the claimant and the decision of the board are predicated upon the finding or ruling that there existed justifiable cause under G. L. (Ter. Ed.) c. 152, s. 30, for Dr. Blood continuing his treatment

of the employee as a private patient. The insurer seasonably filed requests for findings of fact and rulings of law. These requests are numbered 1 to 20 inclusive. The reviewing board gave requests numbered 1, 2, 3, 4, 7, 8, 9, 10, 12, 14 and 18 but refused the others. The board in this connection specifically found the following facts under said s. 30: On October 7, 1930, at about 7 a.m., Katherine Zombric, a girl twenty-two years of age and single, sustained a very serious scalp injury which arose out of and during the course of her employment when her hair caught in a revolving shaft. Necessity required that she be sent to the nearest hospital for specialized and surgical treatment. The nearest hospital was the St. Anne's Hospital. Dr. G. W. Blood was a member of the staff at the time and had been for a good many years, but he was not on service or duty during the period within which this accident occurred. On account of the exigency, Dr. Blood was called by some one connected with the hospital and responded, *rendering emergency service.* Upon the same day, the employee, through her father, selected Dr. Blood as her physician. Beginning October 20, 1930, the insurance company furnished no other treatment, and after conferring with the insurance company and receiving a letter from it, Dr. J. Newton Shirley, employed by the Arrow Mutual Liability Insurance Company, consulted with Dr. Blood each week while the employee was under his care.

The board further found that in the circumstances disclosed by the evidence—that Dr. Blood came in on the case in the emergency, was asked to continue on the case by the employee's father, together with his correspondence, talk and contact with the insurer or its representative,—there existed justifiable cause under s. 30 for Dr. Blood continuing his treatment of the employee as a private patient; that the treatment rendered was adequate and reasonable and a reasonable fee for the services of the physician is $826.

The insurer contends that there was no evidence to support the finding that "upon the same day, the employee, through her father, selected Dr. Blood as her physician." The contention is met by the inference, as above stated, that the action of the father was ratified by the conduct of the employee. The insurer further contends that there is no evidence to support the finding that "beginning October 20, 1930, the insurance company furnished no other treatment, and after conferring with the insurance company and receiving a letter from them (above quoted) Dr. J. Newton Shirley employed by the Arrow Mutual Liability Insurance Company, consulted with Dr. Blood each week while the employee was under his care." This contention is based on the position that "The question here involved is whether a physician on the staff of such a general public hospital is entitled to compensation from the insurer for his services to a patient in such hospital in the absence of any contract therefor with the insurer." The insurer further contends that the following paragraph in the decision of the Industrial Accident Board that "The Board, under the circumstances disclosed by the evidence—that Dr. Blood came in on the case in the emergency, was asked to continue on the case by the employee's father, together with his correspondence, talk and contact with the insurer or its representative,—find that there existed justifiable cause under section 30 for Dr. Blood continuing his treatment of the employee as a private patient," was unwarranted in fact and in law. This contention rests upon the fact that the employee was sent to the nearest general public hospital for emergency treatment, which was rendered by a physician on the staff of the hospital called by the hospital authorities; that there is no evidence the treatment began as that of a "private patient"; that the treatment continued in the same hospital by the same staff surgeon, and the hospital bills were paid by the insurer. As contended, the treatment did not begin as that of a "private patient," but it was continued as such following the employment of Dr. Blood at noon on the day of the accident. The insurer relies on Allen's Case, 265 Mass. 490. That case is distinguishable in its facts from the instant case in two respects (1) the employee did not select the physician, and (2) there was no emergency or other justifiable cause which gave a right of recovery to the physician for services rendered. *In the case at bar there were both an emergency and a selection of a physician after the employee had recovered consciousness, the exact moment of such selection being immaterial.*

Decree affirmed.

E. Field, (R. H. Field with him,) for the insurer.

W. C. Crosley, for the claimant.

Messrs. Cenedella, Gleason, Clark and Mrs. Tousant—for physician.

Affirmed by Supreme Judicial Court.

Opinion filed March 27, 1935.

DECREE OF THE SUPREME JUDICIAL COURT

CARROLL, J. In this proceeding under the workmen's compensation statute Dr. Spellman, a member of the staff of the hospital to which the employee was taken following his injury, seeks to recover compensation for his services as a physician in the treatment of the employee at the hospital. The employee was paid the compensation due him under the statute and the insurer paid the hospital bill amounting to $63.00.

The Industrial Accident Board found that the injured employee consulted a doctor, who bandaged his hand; that he then went to St. Elizabeth's Hospital where a nurse asked him if he had a doctor, and he said he knew no doctor at the hospital; *that the nurse "suggested the name of Dr. Spellman and he said that Dr. Spellman would be all right."* The board further found that the employee did not choose a physician as the statute con-

templated, that the insurer fulfilled the obligation placed upon it by the statute and furnished adequate and reasonable hospital services to the employee, and denied the claim of the physician for services in the care of the employee. In the Superior Court a decree was entered for Dr. Spellman in the sum of $45. The insurer appealed.

G. L. c. 152, s. 30, so far as material, provides that during the first two weeks after the injury the insurer is to furnish adequate and reasonable medical and hospital services; that the employee may select his own physician or in case of emergency a physician other than the one provided may be called to treat the patient at the expense of the insurer, subject to the approval of the department. As we construe the findings of the Board, the insurer complied with the statute and did all that was required of it—it furnished the employee with adequate and reasonable medical and hospital services. Physicians as well as nurses are generally expected to be in attendance at a public hospital. A patient who has been taken to such an institution if he has no physician of his own to treat him naturally expects that he will receive treatment from someone on the staff. When an injured employee under the compensation act goes to such a hospital and does not select a physician, the payment to the hospital of its charges includes the expenses of nurses and physicians, and the insurer is not required to pay the physician, who is a member of the staff for his services.

The Board found as a fact that the employee did not choose a physician. The finding was warranted. *The employee was at the hospital for treatment. His response to the nurse's suggestion concerning Dr. Spellman was not a selection of a physician under the statute.* The employee was willing to accept such facilities as were offered, including nurses and physicians; *he accepted the suggestion of the nurse, but did not make such a selection as the statute contemplates when a patient selects a physician other than the one provided by the insurer.*

There was no emergency which entitled the doctor to compensation under the statute. In case of an emergency a doctor called to treat a patient may recover compensation, but this is subject to the approval of the department and in the case at bar there was no such approval.

Decree reversed.

Opinion filed: January 4, 1929. Decree to be entered for the insurer.

IDENTIFICATION OF CRIMINALS

According to current reports, Dr. Carleton Simon, former Deputy Police Commissioner of New York City, in association with Dr. Isador Goldstein, Ophthalmologist at Mount Sinai Hospital, New York, has after a year's study of the method of measuring the network of structures in the retina of the human eye, devised a system which supplants other methods of identification.

Criminologists have found that chemicals and surgery may invalidate fingerprints and physical peculiarities of suspects, as has been found in some important cases.

The Simon method employs a retinal camera which records the distances of branching blood vessels from the optic nerve through a meshed screen.

It is claimed that anyone can learn how to use the system in two hours and that medical knowledge is not required.

The destruction of finger marks and blemishes has aided criminals in several cases. Enemies of society will not be likely to have their eyes removed.

THE REGISTRATION OF OSTEOPATHS IN GREAT BRITAIN

The Committee of the House of Lords (England) has recommended that the bill for the registration of osteopaths be not further proceeded with.

This action is the result of an intimation by the supporters of the measure that the bill, if enacted, would place osteopaths on a par with doctors, and also because further consideration be given to the scientific basis of osteopathy.

The Committee also reported that "no definition of osteopathy had emerged which satisfactorily differentiated the osteopathic sphere of activity".

There are reported to be from two thousand to three thousand osteopathic practitioners in Britain of whom only about one hundred and seventy can claim to be qualified.—Digest of a report in the *New York Times*, July 21, 1935.

NOTICES

REMOVAL

HAROLD BEECHER HARRIS, M.D., announces the removal of his office to 465 Columbia Road, Dorchester, Massachusetts.

LAWRENCE CANCER CLINIC

Established 1928

Lawrence, Mass.,
July 20, 1935.

To the Physicians of the North Half of Essex County:

Dear Doctor:

The regular Lawrence Cancer Clinic, to be held at Lawrence General Hospital, 1 Garden Street, Lawrence, upon Tuesday, August 6, at 10:00 A.M., will be a Demonstration Clinic with Channing C. Simmons, M.D., of Boston, Surgeon-in-Chief to the Collis P. Huntington Memorial Hospital, and member of the Cancer Commission of Harvard University, Boston, present as consultant. You are invited to accompany any of your patients whom you desire shall have this service, or to send them with a note, and a report will be returned to you. This

service is gratis. Your attendance at the Clinic is always welcome.

This Clinic is endorsed by the Committee on Postgraduate Instruction of the Massachusetts Medical Society.

Committee:

ROY V. BAKETEL, M.D.,
CHAS. J. BURGESS, M.D.,
FRED'K D. MCALLISTER, M.D.,
JOHN J. MCARDLE, M.D.,
HARRY H. NEVERS, M.D.,
THOS. V. UNIAC, M.D.,
J. FORREST BURNHAM, M.D., *Chairman.*

CITY OF NEW YORK, MUNICIPAL CIVIL SERVICE COMMISSION

This Commission will in the near future conduct an examination for the position of Associate Director of the Bacteriological Laboratories, Health Department.

The salary of the position is $6,000 per annum and the duties of the incumbent will be to assist the director of the laboratories in the executive supervision of one of the largest and most important laboratories of its kind in this country.

WM. H. ALLEN, *Secretary.*

Municipal Building, Manhattan,
Centre and Chambers Streets, Fourteenth Floor.

NOTICE OF MEETING

ELEVENTH CLINICAL CONGRESS OF THE CONNECTICUT STATE MEDICAL SOCIETY

NEW HAVEN, SEPTEMBER 17, 18, 19, 1935

The registration fee for the 1935 Clinical Congress will be $2.00. Luncheons are not included in this fee, but will be available at a low cost.

More than 600 physicians from 9 states attended the 1934 Clinical Congress.

To increase the value of the Congress, a Commercial Exhibit of some 25 carefully chosen exhibitors has been added this year. The Exhibit will be held in the new Tompkins East I of the New Haven Hospital and will be open throughout the Congress.

Afternoon sessions each day will be devoted to demonstrations and round table discussions on the subjects that have been presented at the morning sessions.

All papers presented before the Congress will be abstracted in the October issue of the *Yale Journal of Biology and Medicine.*

Free parking of automobiles for members of the Congress will be available near the meeting place. Continuous telephone service will be maintained so that members can be reached at any time by calling New Haven 5-1161, Clinical Congress extension.

Early registration will facilitate the work of the Committee on Arrangements. If you expect to attend the congress, please send your check for $2.00, made payable to the Connecticut Clinical Congress. Names and addresses of all who register before August 25 will be published in the final program to be distributed about September 1. Complete program may be secured by addressing:

Creighton Barker, M.D.
Chairman of the Committee on Publicity and Registration
129 Whitney Avenue, New Haven, Connecticut.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, AUGUST 5, 1935

Wednesday, August 7—

†12 M. Clinico-Pathological Conference. Children's Hospital.

Saturday, August 10—

•10-12. Staff rounds at the Peter Bent Brigham Hospital.

•Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

August 6—Lawrence Cancer Clinic. See page 243.

August 29 - September 5—Latin American Congress of Physical Therapy, X-Ray and Radium. For information address Dr. Madge C. L. McGuiness, 1211 Madison Avenue, New York City.

September 5, 6, 7—American Congress of Physical Therapy will meet at the Hotel Kansas Citian, Kansas City, Missouri. Program and circular of information may be secured by addressing: American Congress of Physical Therapy, 30 North Michigan Avenue, Chicago, Illinois.

September 17, 18, 19—Eleventh Clinical Congress of the Connecticut State Medical Society. See notice elsewhere on this page.

October 7-10—American Public Health Association will meet in Milwaukee, Wisconsin. For information address the American Public Health Association, 50 West 50th Street, New York City.

October 21 - November 2—1935 Graduate Fortnight of the New York Academy of Medicine. See page 898, issue of May 9.

October 28 - November 1—The Twenty-Fifth Clinical Congress of the American College of Surgeons. See page 1065, issue of May 30.

BOOK REVIEW

The Modern Method of Birth Control. Thurston S. Welton. 168 pp. New York: Walter J. Black, Inc. $3.00.

This little book gives a clear explanation of the modern concept of the physiology of reproduction. It is, however, merely another paraphrasing of the work previously published by Ogino and Knaus. It does not differ essentially from Lutz's book "The Rhythm" except in the use of a series of diagrammatic charts, of which there are sixty-seven, together with a celluloid calendar wheel to be used with them. Actually there are only seventy-four pages of text in addition to the short explanations that go with the charts. To your reviewer the use of these charts seems to add an unnecessary complexity to what is probably going to be a very satisfactory method of birth control. As has been said, in reviewing other books on this same subject, to date we lack sufficient information to give this advice in cases of serious illness. Another objection to this book is its price. Three dollars seems a rather large amount for the information given.